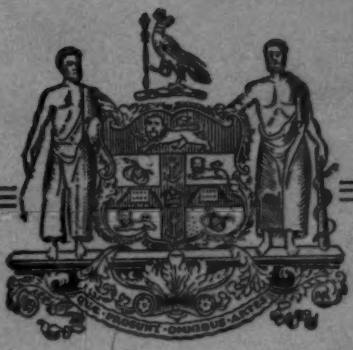

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VOLUME 29 AUGUST 1961 No. 2

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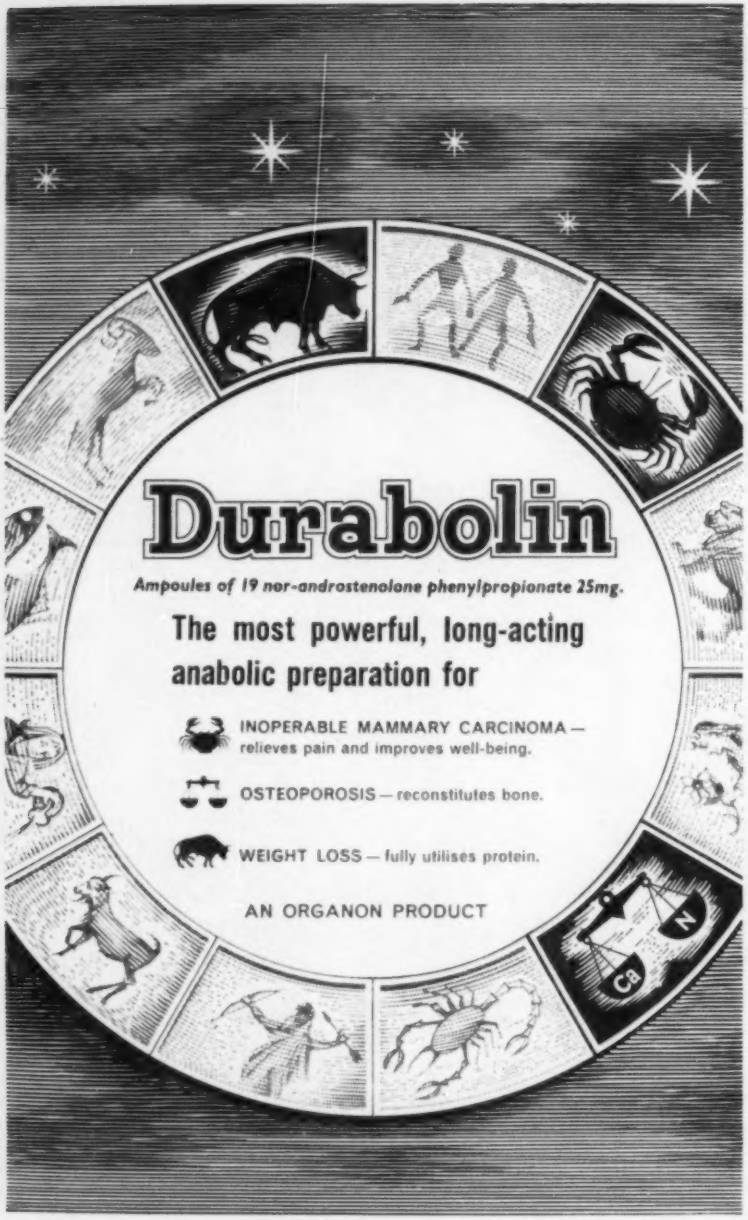
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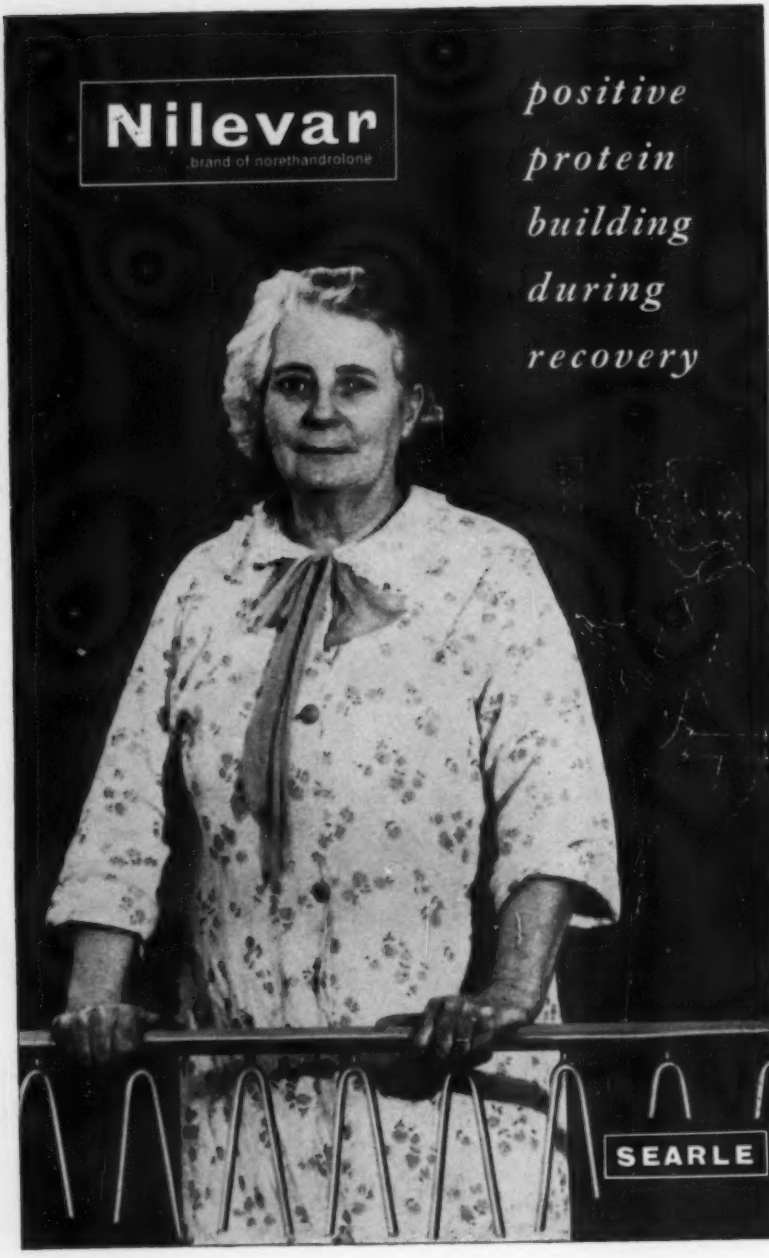
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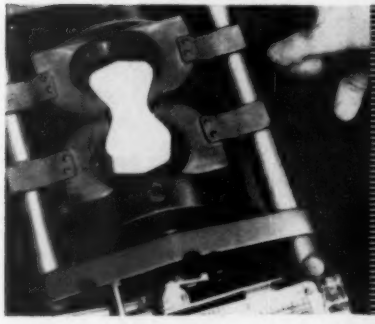

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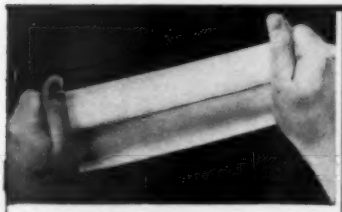

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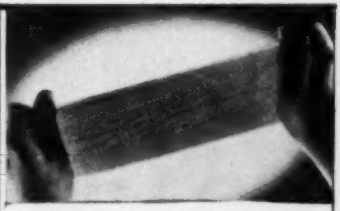

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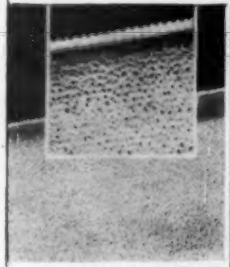

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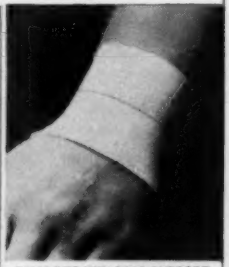

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CONTENTS

AUGUST 1961

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ACTIVATION OF THE RECORD OF HUMAN EXPERIENCE

Summary of the Lister Oration delivered at the Royal College of Surgeons of England*

on

27th April 1961

by

Professor Wilder Penfield, O.M., C.M.G., M.D., D.Sc., Hon. F.R.C.S.

Honorary Consultant to the Montreal Neurological Institute of McGill University

IN 1949 GEOFFREY JEFFERSON stood where I stand to-day to accept the Lister medal in this venerable College and to deliver the Memorial Address. And now we mourn his recent death. He was a most distinguished surgeon, a whimsical, lovable friend. Sir Geoffrey took as the title of his oration, "The Mind of Mechanical Man," and his closing words were these:

"I end by ranging myself with the humanist Shakespeare rather than the mechanists, recalling Hamlet's lines:

"'What a piece of work is man! How noble in reason! How infinite in faculty; in form and moving how express and admirable! in action how like an angel! in apprehension how like a god!'"

Yes, what a piece of work is man. He has created Science and the Machine. But man, the creature, is so much more than anything he will ever create. What computer, past, present or future, could do what Lister did and be what Lister was? What Lister did will never be forgotten by those who practise medicine, least of all by surgeons. But what he was may fade from view. Let me look back then for a moment.

On finishing classical studies at University College, Joseph Lister, a tall handsome young Quaker who had hoped to become a surgeon from early childhood, was filled with misgivings about himself and life. He had a way of stuttering when tired or embarrassed. One biographer, who considered it a family misfortune, pointed out that he had not inherited his mother's beautiful, long, artistic fingers! Instead, his hands were ugly, square, thick, with short fingers—the hands in fact of an artisan, like those of the speaker to-night—like those perhaps of many of the surgeons who have stood here before me. We are artisans first and last.

After his classical studies, Lister took a year out for reflection and reading. Then he entered medicine; but when he had finished the prescribed courses, he turned away a second time, refusing the opportunity to practise surgery, appalled by its failures. He devoted himself, instead, to physiology and pathology for a time. Eventually he entered surgery, moving to Edinburgh, then to Glasgow and back to Edinburgh.

At last he returned to London and with him, from Scotland, came his house surgeon, Watson Cheyne, red-bearded like an ancient Viking, and

---

* The full text is to be published in *Brain*.

his senior clerk, John Stewart, a tall black-bearded athletic Canadian from Nova Scotia, a youth very like Lister in manner and appearance it was said. Stewart was to give the first Listerian Lecture before the Canadian Medical Association in Ottawa in 1924. And Sir William Watson Cheyne was to receive the Lister Medal and inaugurate this lectureship here in the same year.

Lister, as Cheyne described him, was a man who gave to compassion the first claim on his attention, as any physician should. In the afternoon of a routine day, he would often return to the hospital for a second visit to see that all went well with his plans. He might stop at a bed to comfort one who lay there in pain or discouragement and perhaps to change the surgical dressing. Then, continuing on his way preoccupied, come home to a consulting room filled with restless patients, patients who could pay, and doctors pacing the floor resentfully.

He was always courteous, calm, reflective; pursuing the unrevealed, despite the anxious and demanding currents of a surgeon's life. In this, he was like John Hunter, who also had a slight stammer from childhood, but different too, very different. Instead of collecting materials, as Hunter did for this great museum, Lister began by publishing one case of bony exostosis removed by his chief, Professor Syme. In it he analysed the biological process of cartilage formation and bone growth. He reported one single case of carbuncle, just one, taking it as a text for a pathological enquiry into underlying action and reaction. At 30 he discussed before the Royal Society the initial stages of inflammation.

It has been said that Lister and Hunter could never have been friends. But I like to think otherwise, believing in the kinship of true greatness. They showed surgeons how to be more than craftsmen, how to escape from what might be called barber-mindedness, taught them how to turn their thoughts to science and discuss it openly even in the presence of physicians!

Lord Lister died in 1912. Here are phrases from a letter written by a Canadian, Sir William Osler, then Regius Professor of Medicine at Oxford:

" I have just come from the Abbey service—the greatest Englishman of his generation—the most splendid tribute ever paid to our profession—England's tribute of heart and hand."

Such a man as Lister was " must give us pause," for this is God's creation. No man-made machine will equal him, no science ever quite explain. " What a piece of work is man . . . in apprehension how like a god! "

ACTIVATION OF THE RECORD OF HUMAN EXPERIENCE

Realizing that his own biography was inevitable, Lord Lister asked that it should be " a simple record of what he had done for Science and Surgery". I suppose he would want the lecturer in this triennial ceremony to talk simply of something he had done.

In the course of operations on the brain, I have stumbled on some fascinating findings. I must confess that it was usually more by accident than by design. But chance observations, carefully recorded and filed and integrated with the collateral observations of scientific associates, may prove to be a rich mine for subsequent study. The science of surgery is like that, and when it comes to awards who can be quite sure that the one

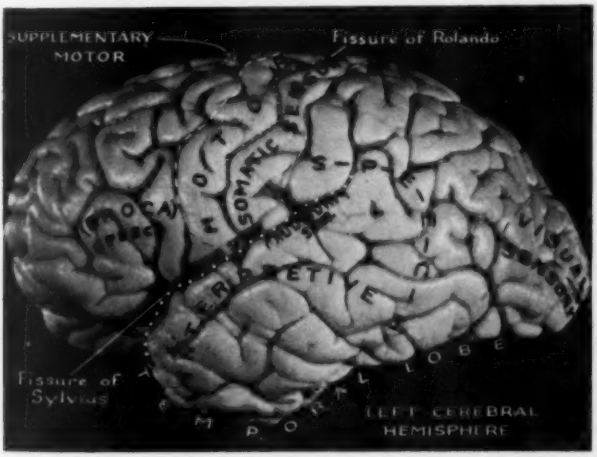

Fig. 1. Left cerebral hemisphere showing areas devoted to speech, sensation, voluntary movement. The area of cortex from which electrical stimulation produces experiential " flash backs " and interpretive signals is labelled interpretive. The auditory sensory area of the cortex is beneath the area indicated; that is on the anterior transverse gyrus of Heschl, but hidden here within the fissure of Sylvius.

who carries the medal away is he who deserves it most? To-night, instead of me, it might well have gone to one of my associates—to Jasper for his neurophysiology, to McRae for his neuroradiology or to Rasmussen, my surgical colleague in this work, for his critical counsel and his skill.

Perhaps the most important group of observations that I have made during an exciting career as neurosurgeon is the one I shall discuss to-night. In any case, this group still calls for the summary and analysis which is possible now, thanks to an exhaustive review carried out by my associate, Dr. Phanor Perot.

Electrical stimulation of the human brain, while patients are conscious, has sometimes activated the neuronal record of past experience. While the surgeon's stimulating electrode is kept in place at some point in the temporal cortex, a secondary stream of consciousness moves through the patient's mind. It is an awareness of things as they were in some previous strip of time. Action goes forward at the original tempo; he is aware of those things to which he paid attention then, and yet he is also aware of the present. When the electrode is withdrawn from the temporal cortex, that experiential review usually stops instantly. Often, when the electrode is replaced at approximately the same spot a minute later, it happens that the stream of past consciousness is caused to flow again through the patient's mind, beginning at the same moment in past time.

During the past hundred years, knowledge of the action of the human brain has come to neurophysiologists gradually. With each forward step, there was recognition of some new partially separable unit of function within the brain. There are areas of cortex and of brainstem, with their connecting neurone circuits, which are devoted to particular functions—sensory, motor, psychical.

The area involved in each functional transaction was often recognized most easily in the cerebral cortex. The first localization was that of speech in a comparatively discrete portion of cortex on one hemisphere, the dominant left hemisphere (Fig. 1); next, voluntary motor control in the precentral gyrus of both sides. After that, visual sensation, auditory sensation and discriminatory somatic sensation, each in well-defined areas: motor cortex, sensory cortex and speech cortex. It must be kept clearly in mind, of course, that these areas could in no sense act independently of subcortical connections.

The newly recognized areas, from which this record of past experience is accessible to the electrode, can be outlined now. We have proposed, therefore, to call the area *interpretive cortex*. The reason for the name will appear later. It covers the surface of the temporal lobes adjacent to the auditory sensory and the visual sensory areas, and, on the left side, adjacent to the temporal speech areas but not overlapping them. Thus it is not the same in extent on the dominant and non-dominant sides. On the non-dominant side it extends over the area that might have been devoted to speech.

The operative and clinical procedures that have made this present summary and analysis possible have all been reported in earlier publications. The operation of osteoplastic temporal craniotomy and an assessment of the therapeutic results of partial temporal lobectomy, as a treatment for focal epilepsy, were described here in the Hunterian Lecture

of July 1953, before this College.* Therefore, I shall pass on, as directly as possible, to consider what light these unexpected experiential responses to stimulation throw on brain physiology.

In preparation for this study Dr. Phanor Perot and I have re-examined the total experience in the Montreal Neurological Institute, 1,288 craniotomies on 1,132 patients in the treatment of focal epilepsy carried out under local anaesthesia (Table I). Experiential responses were produced by stimulation during operations on 40 patients. In addition to this, there were 53 patients who described involuntary recall of previous experience during their minor epileptic attacks. That is to say the epileptic discharge which was occurring locally in the temporal lobe produced this recall. Study of the details of these 93 examples of activation of the neuronal record of experience brings us to certain conclusions.

TABLE I

CASE MATERIAL

Patients in stimulation series	1,132
Patients with temporal lobe stimulation ..	520
Right side	248
Left side	272
Patients with experiential responses ..	40 (7.7%)
Patients with ictal experiential hallucinations	53 (10%)

The experiences recalled are chiefly auditory or visual, or else they are combined auditory and visual. Figure 2 shows the points from which all types of experiential response occurred. As suggested in Figure 3, the purely auditory experiences occur on the superior temporal convolution (lateral and superior surfaces), while the purely visual may occur there, but more often in the non-dominant temporal cortex. None fell in the active speech area of the dominant hemisphere and none in other lobes of the brain.

Curiously enough, there are no examples of recall of a time when the individual was devoting himself to his own action—no example of a time of eating, or of sexual experience, or of concentrated reasoning, or running a race, or singing, or playing the piano. There are many examples of hearing complicated music, sometimes accompanied by seeing the scene in which the music was originally heard and feeling the emotion that it produced.

It is not in the interpretive cortex that the actual record of experience is located. That is elsewhere in the brain, in a place perhaps where all experience may be recorded by retained patterns of the previous passage of nerve impulses. These are the neurone patterns that preserve the past. They hold the pathway of passage of electrical impulse in a facilitated sequence, which can be reactivated as though an electric current were being passed through a tape recorder. Thus this conscious experience

* *The British Journal of Surgery*, 1954, **41**, 168.

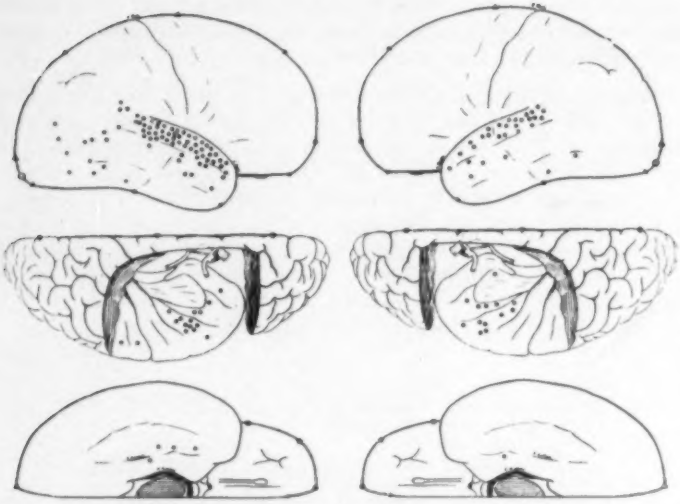

Fig. 2. The hemispheres of the brain, showing lateral, superior, and inferior surfaces. In the middle pair of drawings, the frontal and parietal areas have been partly removed so as to expose the superior surfaces of the temporal lobes. The solid dots show all the points from which electrical stimulation produced experiential responses. No responses of this type were produced by such stimulations of the grey matter of other lobes.

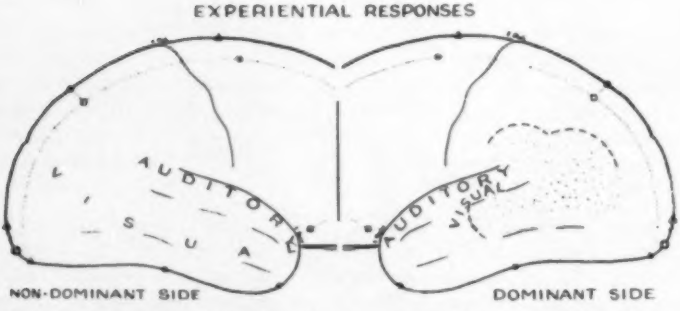

Fig. 3. Right and left hemispheres showing the general location of points producing experiential responses which were chiefly auditory. Also those which were visual in character. Experiential recall which was both auditory and visual had its location chiefly on the superior temporal convolution. The dotted zone indicates the posterior speech area as outlined by production of aphasia by electrical interference, as worked out by Penfield and Roberts (1959).

is on record in the brain though usually out of reach of voluntary recall. The interpretive cortex has direct connection with that vast file of past experience, especially the experiences during which attention was turned to things seen and heard.

TABLE II

PSYCHICAL RESPONSES TO ELECTRICAL STIMULATION OF INTERPRETIVE AREAS OF CORTEX

A. *Experiential Flash-Back:* Random re-enactment of a conscious sequence from the patient's past.

B. *Interpretive Signalling:* Production of sudden interpretations of the present experience, such as familiar, strange, fearful, coming nearer, going away, etc.

It may be recalled that stimulation of the brain of conscious patients has from time to time produced psychical responses of two types, as illustrated in Table II. The significance of these findings in terms of brain function may be as follows:

With each new current experience similar previous experiences, if there are any, are selected instantly from that neuronal file. They are automatically activated by the brain's own system and thus a signal of interpretation is flashed into consciousness. The signal may be one of familiarity, a notice that here is what was once known to you, made suddenly available so you can add to this cross-index whatever may be new and reinforce the record of what was old.

The new experience may have to do with a man or a woman or a place—something seen and heard years ago. Yet the interpretive signal warns you that you have seen and heard this before. On the other hand, the signal may be one of fear rather than familiarity. Things may be coming at you or going away. As a result you may be on your guard or may take evasive action before you "have time to think". The signal tells you automatically. Subconscious interpretations of present experience are only possible if past experience is made available in detail. This is one of the mechanisms of memory. Some reactions called conditioned reflexes when seen in the dog must be recognized as these interpretations in man.

I have described to you a body of evidence that has come to us while studying the problems of epileptic patients, and during operations designed to cure temporal lobe epilepsy. It was epilepsy that guided Hippocrates to his remarkable grasp of brain function. Jackson made his shrewd guesses by watching local fits. Epilepsy is pointing the way still, no doubt, whispering the answers. But it is hard to interpret the whisper, difficult to comprehend the functional meaning of the "dreamy states", and to express that meaning in terms of mind and brain.

In conclusion we have watched epileptic and electrical irritation activate the neuronal mechanism of subconscious interpretation of the present. We have seen it re-activate the record of the stream of consciousness and

have concluded that, functionally, the two mechanisms are closely interrelated. This is only a part of the basis of what men call memory, a small step towards greater understanding of the master organ, the organ that makes man what he is and puts such power in his hands for good or bad.

" What a piece of work is man " that he can be what Lister was, and do what Shakespeare did! " How infinite in faculty; in form and moving how express and admirable! " And yet how blind he seems, how bent on self-destruction! Surely the most important work in the world to-day is the study of man by man.

ELECTION TO THE COUNCIL

On Thursday, 6th July, Professor C. A. Wells, Professor R. Milnes Walker, and Professor Ian Aird, were re-elected, and Mr. N. L. Capener and Mr. E. G. Muir were elected Members of the Council of the College.

The result of the Poll was as follows:

Elected	*Votes*
Ian AIRD (Postgraduate Medical School of London)	1,207
Robert Milnes WALKER (Royal Infirmary, Bristol)	1,178
Charles Alexander WELLS (Royal Infirmary, Liverpool)	1,033
Norman Leslie CAPENER (Royal Devon and Exeter Hospital)	712
Edward Grainger MUIR (King's College Hospital)	663
Not elected	
David Howard PATEY (Middlesex Hospital)	638
Alec William BADENOCH (St. Bartholomew's Hospital)	602
Rodney SMITH (St. George's Hospital)	578
Alexander Michael BOYD (Royal Infirmary, Manchester)	573
David Napier MATTHEWS, O.B.E. (University College Hospital)	548
David TREVOR (Charing Cross Hospital)	421
Thomas Geraint Illtyd JAMES (Central Middlesex Hospital)	407
Harvey JACKSON (National Hospital and St. Thomas's Hospital)	371
George QVIST (Royal Free Hospital)	363
Robert Wallace NEVIN, T.D. (St. Thomas's Hospital)	351
Ronald William RAVEN, O.B.E., T.D. (Royal Marsden Hospital)	350
Edward Clive Barber BUTLER (The London Hospital)	303
Stanley Osborn AYLETT, M.B.E. (Gordon Hospital, Westminster Group Hospital)	254
Richard Arthur MOGG, V.R.D. (Royal Infirmary, Cardiff)	230
Donald Spiers Monteagle BARLOW (Southend and Luton Groups of Hospitals)	222
Clifford David Phillips JONES, M.B.E. (Sheffield Royal Infirmary)	202
Thomas NICOL (King's College, London)	158

In all 2,640 Fellows voted, and in addition 21 votes were found to be invalid.

GALLSTONES

An Introduction to Research into Causes and Remarks on Some Problems in Treatment

Lecture delivered at the Royal College of Surgeons of England
on
4th October 1960

by

A. J. Harding Rains, M.S., F.R.C.S.

Professor of Surgery (University of London), Charing Cross Hospital Medical School

THE NUMBER OF stones found in the gallbladder varies between one and thousands, and even in the bile ducts they may be single or multiple. However, it is usually one stone amongst many which is responsible for the suffering of the patient and for the specific clinical features and secondary pathological effects.

For the immediate needs of the patient we require a knowledge of methods of diagnosis, detection and removal. There might appear to be little practical importance in gaining a knowledge of the theories of the causation of stones, and, moreover, research leading to the ultimate discovery of the cause will favour preventive medicine at the expense of surgery. Yet, in spite of this, one should welcome and play a part in such progress, hoping that a dissemination of existing knowledge will provide many with a stimulus to thought and research.

Thus, before considering certain clinical aspects a short review of research into causes is presented.

Types of stones

(*a*) "*Mixed*" *stones.* These are the commonest. Single or multiple, they are composed of cholesterol and bile pigment with proteins, carbohydrate, calcium, iron and phosphorus (see chemical analysis below). Outwardly they may have a covering of whitish cholesterol or brownish pigment. The presence and pressure of adjacent stones tightly packed within the gallbladder causes faceting and makes them polyhedral rather than spherical in shape. On cross-section one may see concentric laminae of cholesterol crystals or amorphous pigment, and at the centre a dark softish "nucleus" or even a crevice or space.

(*b*) *The "pure" cholesterol stone.* The single "pure" cholesterol stone, cholesterol solitaire, is uncommon (Fig. 1). Analysis reveals that it is not composed solely of cholesterol, for protein, fat, carbohydrate and even pigment may be present. It is light in weight as well as in colour, is rather "soapy" to feel and has a granular surface. The radiating crystals of cholesterol are seen on cross-section, and at the centre a small amount of bile staining may be present.

Multiple cholesterol stones, resembling hundreds of small pearls, are not pure. Under the pearl-like covering a crumbling yellow or brownish substance is found.

(*c*) *The pigment stones.* These also are not as common as the mixed stone (found in seven out of 57 cases recently examined). They are multiple, even into thousands, and are suspended in dark bile or black

Fig. 1. The cholesterol solitaire stone.

sludge. There is a gradation in size from 5 mm. down to the sludge particles. Irregular in shape, with sharpish edges and points, they are glistening black, hard and compact. Occasionally they are feathery and coral-like and may be lying in normal-looking bile (Fig. 2). More rarely they are single, of larger size (about 1 cm.), rounded with a slightly rough surface and are of hard consistency except perhaps in the centre.

Pathways of research into the causes of stone formation

(*a*) *Chemical Analysis.* Most of the chemical constituents of stones have been known for a long time (Thomson, 1833). Naunyn (1892) was concerned with the occurrence of cholesterol, bile pigment, calcium and carbonate in the different types of stones and with the appearance of the nucleus. He believed that the common gallstone with a pultaceous or crumbling centre is formed in the first place by a soft mass onto the surface of which cholesterol is deposited. Crystallization of cholesterol inwards towards the centre of the mass then follows (cholesterinization). Cholesterol was presumed to come from bile or through the wall of the gallbladder. On the other hand those mixed stones with hard centres of pigment (in addition to pure pigment stones) were supposed to be developed from a nucleus of calcium bilirubinate.

Rovsing (1924) said that pigment could be seen in the centre of all stones and postulated that they must originate as a small concretion of calcium bilirubinate, adding that such a nucleus originates in the bile canaliculi of the liver as a result of disease in this organ and is transported in the bile to the gallbladder.

Recently, we (Rains, Barson, Crawford and Shrewsbury—1960) have examined and analysed stones from 57 patients. Macroscopical in-

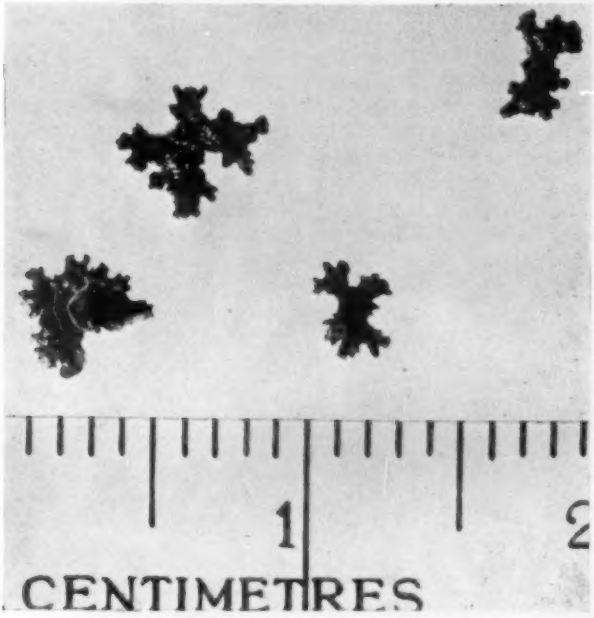

Fig. 2. Small coral-like bile pigment stones.

spection confirms that pigment is nearly always seen in the centre of stones (Figure 1 was the only exception in this series).

TABLE I

ANALYSIS OF 57 SPECIMENS OF GALLSTONES

Substance	*Number of whole stones in which substance is present* (%)	*Centres of stones in which substance is present* (%)
Protein	49 (86)	37 (65)
Cholesterol	47 (82)	43 (75)
Bile pigment	45 (79)	42 (74)
Carbonate	46 (81)	33 (58)
Calcium	20 (35)	14 (25)
Phosphorus	14 (25)	11 (20)
Iron	7 (12)	5 (10)

Analysis (Table I) does not confirm either this or Rovsing's theory, but it must be stressed that nuclei found negative to tests for pigment did in fact look pigmented. Hammarsten (1914) referred to this pigment as bilihumin, but since that day we have no further information. Table I shows that protein and carbohydrate should command just as much interest as cholesterol and pigment, especially with reference to the result of bacteriological study.

(*b*) *Bacteriological study*. The nuclei of other calculi or concrements found in Nature often contain extraneous matter. Raphael Dubois is said to have discovered in 1901 the presence of a parasite in the centre of a pearl and he described the pearl as the brilliant sarcophagus of a worm. It was perhaps with this in mind that Moynihan described the gallstone as a " tombstone erected in the memory of dead bacteria " for by that time the cause of gallstones was believed to be due to infection of the gallbladder, and that desquamated epithelium, abnormal protein and dead bacteria act as the nucleus. In the 1890's Welch and Cushing had shown typhoid and coliform organisms in stones and gallbladders. Richardson (1898) noted a clumping of typhoid bacilli in bile, like a giant Widal reaction, and suggested that this phenomenon pointed to a bacterial cause.

With the passing of typhoid fever as a common illness and the emergence of the streptococcus in the field of medical problems between 1914 and 1939 we see a corresponding change in the organism of special interest.

Rosenow (1914) found streptococci in the centre of 25 out of 30 stones; bacillus coli in five; bacillus typhosus in one. Illingworth (1927) found seven out of 23 stones infected (two with streptococci, three with bacillus coli and two with staphylococci). The wall of the gallbladder was examined and Rosenow found streptococci in 21 and bacillus coli in 10 out of 29 specimens; Illingworth examined 100, finding streptococci in 34, bacillus coli in three and staphylococci in two.

Rosenow, and Wilkie (1927), showed that stones could be formed experimentally in rabbits by intravenous injection of these organisms. Gilbert and Fournier (1897) had demonstrated this phenomenon with attenuated cultures of typhoid bacilli. Years later we find Martensson (1941) making a comprehensive study of biliary disease and discovering what he called a " typical " bacillus in some stones. Inoculation of attenuated organisms in rabbits produced stones experimentally. More recently we (Rains *et al.*, 1960) began to re-examine this problem. In addition to the chemical analysis of stones from 57 cases, smears and cultures were made of each central portion (nucleus area). In 30 cases an actinomycete was cultured. By agglutination absorption tests this organism has been shown to be distinct from the actinomyces bovis. In addition to these findings, bacillus coli was found in four stones, streptococcus in two and a staphylococcus in eight.

A comparison of the chemical findings between those stones with actinomyces and those without failed to signpost a new direction for

research in chemical analysis, but more work is required on the serology of this organism and its possible relationship to the actinomyces found in dental tartar (Gallippe, 1886; Naeslund, 1925). This may be due to a specific chemical activity, for it appears to contain phosphatase.

No conclusion must be drawn yet from such bacteriological findings. The organisms we find may be mere commensals. Stones which are negative to culture need equal consideration to those which are positive. The riddle of the tombstone is still unsolved.

(*c*) *Investigations concerning metabolism and diet.* Increased excretion of bile pigment or cholesterol appears to be associated with stone formation. Pigment stones (Fig. 2) frequently occur in cases of haemolytic anaemia—whether congenital or acquired—and it is presumed that these form in the bile canaliculi in the liver and that their first appearance within the gallbladder is incidental to the ebb and flow of bile into that viscus.

It has commonly been supposed that a state of hypercholesteraemia might predispose to gallstone formation through an increase of cholesterol in the bile. The raised serum cholesterol associated with pregnancy was thought to be a cause of the frequency with which gallstones are found in multipara. Experimental work of Ghose (1932), Illingworth (1929) and Patey (1934) shows that prolonged feeding of large amounts of cholesterol to rabbits will lead to a cholesterosis of the gallbladder especially if infection is introduced at the same time. Cholesterosis is not often seen in the gallbladders one removes and it is likely that, though this is a phenomenon of hypercholesteraemia, it does not play a part in stone formation as we commonly see it. Further, Piper and Orrild (1956), studying a family of 400 persons suffering from hypercholesteraemia, found no increase in the raised incidence of gallstone cases, though atheroma and the acute vascular episodes of coronary and cerebral thrombosis were very greatly increased.

At the opposite end of the dietary scale there is information of equal interest. Dam and Christensen (1952 and 1954), experimenting with dietary deficiencies and avitaminosis in hamsters, were surprised to find gallstones developing in those animals fed on a particularly poor diet. This led to work on factors in the diet which would inhibit stone formation, and certain unhydrogenated fatty acids and the copper ion appeared to be the necessary substances. Many attempts were made to find a specific causal factor in the diet, but no one item could be detected.

One is left with an impression that, whereas hypercholesteraemia may influence the occurrence of cholesterosis and other such deposits in the body, actual gallstone formation (as originally believed) depends upon the presence of an abnormal physico-chemical state of the bile caused by infection of the gallbladder. The effect of diet deficiency in animals requires more study; it may well be that the poor physical state increases the liability to general infection via the skin and mucous membrane.

(*d*) *Physico-chemical studies and comparative phenomena.* In 1858, Rainey wrote a treatise entitled " On the mode of formation of shells in animals, of bone and several other structures by a process of molecular coalescence demonstrable in certain artificially formed products ". He used solutions of a calcium and protein containing substance (gum acacia) and potassium subcarbonate and showed the development of small clear round bodies of calcium carbonate on a slide put into the glass container. The small bodies gradually coalesced into larger concretions. Figure 3 shows the result of a recent confirmatory experiment. Sixty years later, Peyton Rous, McMaster and Drury (1924) met with a similar phenomenon when studying the sediment formed on the side of glass tubes draining bile in the experimental animals—calling such calcium carbonate

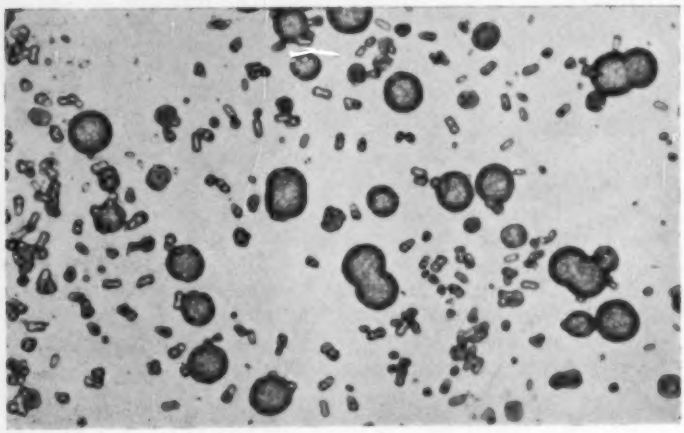

Fig. 3. Coalescence of micro-spheroliths of calcium carbonate.

or bilirubin nuclei " spheroliths ". They claim to have shown that carbonate spheroliths not infrequently serve as the centres for the formation of secondary stones of carbonate and cholesterol, this being influenced by the pH changes in the bile. Schade (1910) demonstrated that a similar phenomenon occurs with cholesterol when it is supersaturating a chloroform or oil solution. The soft spheres so precipitated were " guttulate bodies " and Schade indicated (like Rainey (Fig. 4)) how they might coalesce to form a concretion. It is interesting to compare this with Figure 5 of gallstones which exhibit the same phenomenon.

Molecular aggregation. Rainey's " molecular coalescence " was a coalescence of small particles as seen down the microscope and so does not refer to the natural phenomenon of coalescence or aggregation of molecules. This may also play a part in gallstone formation. Our work

(Rains and Crawford, 1953) on such physico-chemical phenomena started in 1950. Interest in the cholesterol/bile salt relationship required the study of the physico-chemical characteristics of bile salts. Measurement of specific conductivity, osmotic co-efficient and surface tension indicated that molecules of bile salts in solution will at certain critical concentrations aggregate to form larger molecular masses which are called " micelles ". Once these micelles are formed the ability of the solution to dissolve cholesterol is increased. Temperature and pH affects the solubility. It is also known that the presence of phospholipids in the bile increase the solubility. When cholesterol does precipitate out as a result

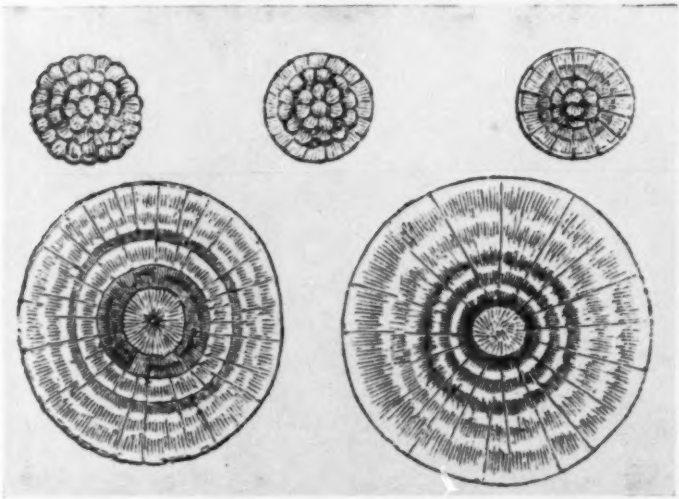

Fig. 4. Rainey's (and Schade's) concept of the formation of concretions and stones. The top left shows the coalescence of spheroliths which, as shown in the other drawings, gradually merge together to form a concretion with concentric laminae and radiating lines of crystallization.

of altering these *in vitro* conditions it does not form a stone, and it would seem that a protein-like substance is necessary to act as a kind of interface.

It is interesting that the term " micelle " is frequently heard nowadays in research into nucleic acid structure and blood-clotting mechanisms, though its originator, McBain (1913), was working with long-chain paraffin salts and he pioneered the work which has given rise to the detergent and polymer industry.

Stasis of bile. Experimental ligation of the cystic duct after inoculation of the gallbladder with attenuated organisms results in cholecystitis. Thus stasis may play a part in providing the necessary environment for

cholesterol precipitation. Imperfect emptying certainly results in layers of bile of different specific gravity filling the gallbladder, and it is at the interface between these layers that spheroliths (as indicated by Rainey's *in*

Fig. 5. Small gallstones formed by the coalscence of smaller stones measuring less than 1 mm.

vitro experiments) may form (Fig. 6). In addition it is obvious that such small stones or calculi would be voided without difficulty or symptoms if emptying were normal, and larger stones could not be formed. The occurrence of stasis must therefore continue to be considered as a factor in gallstone formation and growth.

(*e*) *Is the gallbladder necessary for stone formation?* Stones forming in the hepatic and common bile ducts in cases of congenital absence of the gallbladder suggest that this is not so. In addition there are many examples of multiple stones being found in the common duct many years after satisfactory choledochotomy where it is unlikely that one or more stones had been left behind in the stump of the cystic duct or in the common ducts.

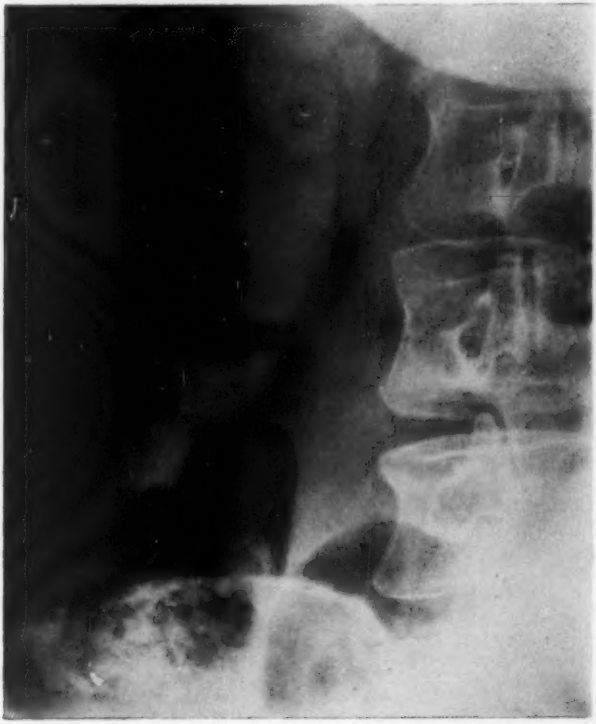

Fig. 6. Cholecystogram which shows a layer of small gallstones at the interface. (This figure does not imply that these stones were formed in this way.)

(*f*) *Foreign bodies.* As with the urinary tract, the presence of a suture can result in stone formation (Homans, 1897). In the biliary system, for example, the stone might form on the remains of sutures used in choledochotomy and cholecystotomy wounds (Fig. 7). The occurrence either supports the infection theory of stone formation or indicates that the surface of the suture provides the necessary interface for cholesterol

precipitation. The exceedingly rare infestation of the ducts by a fluke causes calcification of the walls rather than stone formation within the lumen.

(*g*) *Clinical statistical research.* Horn's paper (1956) is a useful introduction to this kind of investigation. He was interested in testing the validity of the aphorism that gallstones characteristically occur in females who are " fat, forty and fertile ". Clinical and post-mortem material was

Fig. 7. The gallstones, one large and barrel-shaped and the other small and like a ring doughnut, have formed around a nylon suture.

studied. It was found that gallstones increase in frequency with age. In the years following the female climacteric the sex difference diminishes.

From 5,699 autopsy records (3,523 males and 2,176 females) stones were found in 205 males (58.2 per 1,000) and in 287 females (131.9 per 1,000). The age span was 20–89 years. The incidence in the 40–89 year span was:

(*a*) *In males*

40–49 years	26.8	per	1,000
50–59 ,,	50.1	,,	,,
60–69 ,,	56.6	,,	,,
70–79 ,,	116.4	,,	,,
80–89 ,,	217.4	,,	,,

(*b*) *In females*

40–49 years	67.9	per	1,000
50–59 ,,	153.1	,,	,,
60–69 ,,	143.6	,,	,,
70–79 ,,	206.0	,,	,,
80–89 ,,	238.0	,,	,,

Below 50 years, Horn found that stones were more frequent in married women without children than in single women, and more frequent in

married women with children than those without. At ages under 30 years, women with stones were heavier than normal women of a comparable age and height. After 50, differences in weight were no longer apparent. Thus such a study suggests that research into hormone influences on cholesterol metabolism may prove useful. It seems that the

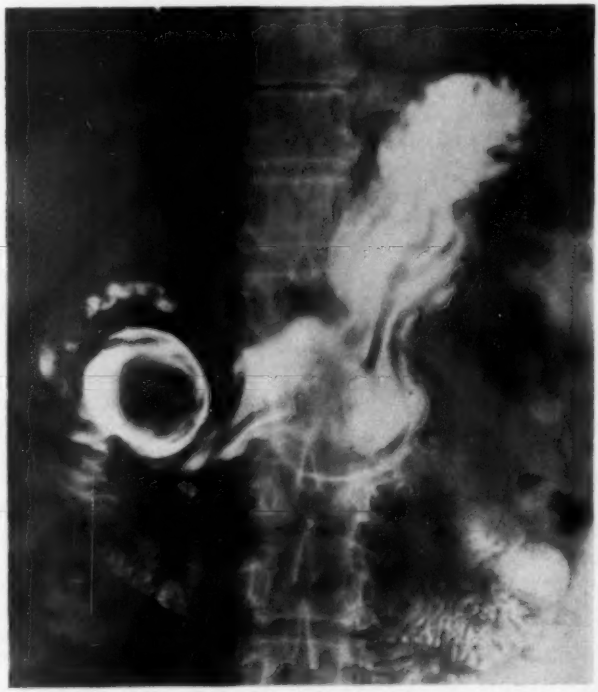

Fig. 8. Barium meal showing large rounded filling defect in duodenum due to a gallstone which has ulcerated through from the gallbladder. Note that the gallbladder and cystic duct is outlined by the barium and that air is present in the common bile and hepatic ducts.

popular aphorism may describe a certain type of patient with stones but not the typical patient. Horn did not compare the incidence of stones in fair-haired with that in dark-haired " fat, forty and fertile " women!

Some remarks on the treatment of biliary stones

The usual clinico-pathological classification is as follows:

Stones in the gallbladder may:

1. Remain and be symptomless (silent or asymptomatic stones).

2. Remain and cause symptoms—chronic cholecystitis—carcinoma of the gallbladder.

3. Pass through the cystic duct.

4. Impact in Hartmann's pouch.

5. Impact in cystic duct.

6. Ulcerate internally into the duodenum (Fig. 8) or colon. Ulcerate externally—through abdominal wall via an abscess.

Stones in the common duct system may either:

1. Pass into the duodenum.

2. Impact in the common bile duct or at the sphincter of Oddi.

3. Impact in an hepatic duct (jaundice if in common hepatic duct or porta hepatis).

4. Be associated with the development of pancreatitis.

Such a clinico-pathological classification does not, I think, portray vividly enough those *clinical situations* with which the patient, the general practitioner and the surgeon usually have to contend. These are:

(1) The accidental discovery of gallstones on plain X-ray (or at operation) for other conditions.

(2) Gallstones diagnosed by contrast X-rays, i.e. cholecystography, for upper abdominal symptoms (which may or may not be strictly referable to the gallbladder).

(3) Acute symptoms referable to the gallbladder and the bile ducts: cholecystitis—empyema of gallbladder—fistula and gallstone ileus—colic—jaundice—pancreatitis.

I wish, firstly, to consider headings (1) and (2) together, and secondly to restrict remarks on (3) to the treatment of acute cholecystitis. The problems of management, operative technique and complications of operations on the common bile duct are frequently dealt with at length in other lectures in this College and any shortened form or résumé would not be very helpful in this lecture.

Treatment Policy. I. On accidental discovery of gallstones by plain X-ray or at operation and also by contrast X-rays (cholecystography) on investigation for upper abdominal symptoms.

Only about 10 per cent. of gallstones contain enough calcium to show up on a plain X-ray, and it is surely a mistake to consider only radio-opaque stones when deciding whether or not gallstones are causing symptoms in a particular patient. Radiolucent stones which show up on cholecystography for upper abdominal symptoms may be just as silent as those which are shown on a plain X-ray. Horn, who has just reminded

us of the frequency of gallstones in the population, also provides us with useful information regarding the percentage of gallstone patients in whom stones are likely to be silent and those in whom stones may be symptomatic: such information is of value in leading us towards a treatment policy.

Horn divided into two groups the 492 autopsy records which revealed the presence of gallstones. There were those which showed that while stones were present, they were not impacted or associated with other gallbladder or bile duct pathology. These were labelled " Asymptomatic Stones ". Into the other group—those with " Symptomatic Stones "—were put cases in which stones were found impacted or there was co-existent biliary pathology. In the fifty-year span (40–89 years) just over half in either sex could be called asymptomatic stones (58 per cent. in females, 55 per cent. in males). Such information inclines one to conservatism in management unless there are definite symptoms or other hazards. Let us consider radio-opaque and then radiolucent stones.

Radio-opaque stones. If such stones are found quite incidentally on X-ray of the abdomen for another suspected or known condition and the symptoms are clearly caused by such a condition, the stones may be considered " silent " and it is customary not to advise operation. However, operation should be considered in cases where large mixed faceted stones are seen, for it is probable that these are filling out a gallbladder now devoid of function. In such circumstances there are the future possibilities of acute cholecystitis and fistula formation into the duodenum (Fig. 8) or colon or even the skin. Added to this is the chance of carcinoma of the gallbladder being present or occurring later (in about 6 per cent. of all gallbladders with stones). The use of a contrast X-ray, e.g. cholecystogram, is therefore justified and if the gallbladder is proved to be non-functioning cholecystectomy should be advised.

Radiolucent stones. In a patient referred for subacute gastro-intestinal symptoms in the upper abdomen it is often difficult to decide which investigation should have priority. If a cholecystogram is done and shows radiolucent stones in a functioning gallbladder, we may wrongly advise cholecystectomy by not realizing that after all these stones are just as " silent " as those radio-opaque stones which are accidentally discovered. As I have intimated, these are the most difficult cases over which to come to a decision, especially as the patient almost certainly is told that gallstones have been found. It is from this group that we are haunted by those who still complain of symptoms after cholecystectomy; for they are still suffering from the condition which caused them to come for advice. Therefore, the point in such circumstances is to concentrate particularly upon the differential diagnosis. This is a fearsome list: hiatus hernia, peptic ulceration, appendicular and colon disease, cardio-respiratory, liver and renal problems. The difficulty is to know what is going to be the next most useful investigation and also to sense when one should stop doing investigations. The best investigation is that of history-taking, to go back

very carefully over the history, focussing sharply onto the description of the symptoms and enquiring for other symptoms suggestive of disease in the other organs or systems. One also has to bear in mind that cholelithiasis is sometimes part of a group of intra-abdominal conditions such as hiatus hernia, duodenal ulcer, chronic appendicitis and diverticulitis. Thus assessment is difficult, but only after a reasonable exclusion of the different diagnoses can cholecystectomy be advised.

If investigation by cholecystogram or intravenous cholangiogram proves definitely that the function of the gallbladder is impaired then, still taking careful note of the possible differential diagnoses, one may more confidently advise cholecystectomy.

If gallstones are found at laparotomy for another condition, cholecystectomy (or cholecystotomy) should not straightway be performed except if it is obvious that the gallbladder pathology is responsible for the symptoms. Multiple operative procedures within the abdomen which add cholecystectomy to the traditional appendicectomy increase the sum of morbidity and mortality.

Treatment Policy. II. Acute cholecystitis

Once stones within the gallbladder become impacted in the cystic duct or in that sigmoid part (Hartmann's pouch) adjacent to the duct, the gallbladder usually becomes isolated from the common duct system. The effect on the gallbladder is said to depend upon the presence or absence of infection (empyema or mucocele respectively). Unfortunately bacteria cannot always be isolated even in the most fulminating cases. However, as far as the patient is concerned, acute cholecystitis (whatever the process may be) requires immediate treatment. As far as operation is concerned, orthodoxy prescribes, rightly I think, a conservative attitude towards the acute gallbladder. Hospitalization is certainly necessary for the severe cases, for those with symptoms of increasing severity, or where the facility for frequent observation and home nursing is difficult and also where the diagnosis is in doubt. Whether admitted to hospital or not, a resolute ten-day course of penicillin and streptomycin or of chloramphenicol or tetracycline may be given even though the indications are not absolute (see (6) below). As the condition subsides, judged by the gradual disappearance of the tenderness below the right ribs, one plans for confirmatory X-ray investigations and cholecystectomy eight to 12 weeks later. However, in a fulminating case where increasing pain, tenderness and pulse imply gangrene of the gallbladder, local peritonitis, abscess formation or a more general spread, an emergency operation should be performed.

Semi-routine emergency cholecystectomy. Except in the mildest cases, emergency cholecystectomy has recently been advocated by many surgeons. The facts to consider are these: (1) the gallbladder is relatively easily removed within the first three days of the onset of the disease;

(2) considerable oedema does not impair but rather assists removal; (3) operability is, however, marred by an increased mortality, for often the patients are acute cardiovascular or respiratory problems at the same time; (4) after three days these problems and the mortality lessen, but one has a little more difficulty with the operation, and so the morbidity may be greater; (5) after 7–10 days the amount of collagen and young fibrous tissue in the area precludes operation unless it is absolutely necessary; (6) if one always adopted the emergency operation policy it would be preferable if antibiotics were *not* used pre-operatively for they tend to increase the density and extent of the fibrous tissue reaction.

Sometimes even with the orthodox conservative policy frequent subacute attacks dictate that an operation should be performed before the usual eight to 12 weeks after the first attack has elapsed.

"*Cholecystectomy versus Cholecystotomy.*" Controversy over the correct operation for stones in the gallbladder has never completely died down since Lawson Tait performed the first successful premeditated operation in 1878. This was the removal of the offending stones (cholecystotomy). In 1882 Langenbuch introduced cholecystectomy. The heading to this paragraph is taken from a letter by Tait in 1885 in which he states the case for cholecystotomy. Cholecystectomy (only three years old) had a mortality of 50 per cent. Tait reported 15 cases of cholecystotomy without a death. However, over a period of thirty years cholecystectomy became the treatment of choice. Cholecystotomy may drain an abscess in the gallbladder and relieve tension, but it will not always enable an impacted stone to be removed. Recurrent abscesses and mucous fistulae emanating from the gallbladder will occur and in such cases cholecystectomy must eventually be performed.

To-day cholecystotomy still has a special place in treatment. In acute cases it is indicated as a "life-saving" procedure, when the patient is quite unfit for a larger exploration yet requires immediate relief from the tense inflamed gallbladder. Cholecystotomy should also be performed when the environment for performance of cholecystectomy is unpropitious (operating theatre and staffing conditions and the degree of training and experience of the surgeon and anaesthetist). Even with such a drainage operation as this is, special care must be taken to prevent seepage of bile and mucus into the general peritoneal cavity by affixing the gallbladder to the peritoneal opening of the drainage route through the abdominal wall.

Non-operative dispersal of biliary stones

Normally, bile will flow through the sphincter of Oddi as soon as food enters the duodenum. With the golden bile from the ducts comes the darker and more concentrated bile which has been stored in the gallbladder between meals. With proper filling and emptying of such a system it is reasonable to believe that biliary stones could not form, but if they did

they would be passed while still small. Olive oil and salines (e.g. magnesium sulphate) will assist emptying, and bile salts taken by mouth will augment the general flow of bile from the liver. Bile salts, particularly those which are conjugated (i.e. sodium taurocholate), have an additional though perhaps theoretical effect. They have those physico-chemical properties which I outlined earlier in this lecture, namely to aid the solubility and excretion of cholesterol. Human biliary stones will dissolve in ox bile or if they are put into the gallbladder of the dog, and it would be expected that an increased excretion of bile salt consequent upon ingestion by mouth might dissolve stones. Further, since lecithin is another factor in dissolving cholesterol this should also be taken. Unfortunately this mixture increases the excretion of cholesterol from the liver so neither solvent is available for dissolving a stone.

Following choledochotomy, gallstone solvents have been given by mouth or introduced down the T-tube in an effort to dissolve sludge or a remaining stone. Ether (Pribram, 1935) has been injected into the T-tube in order to dissolve the cholesterol in such a stone. Any possible good effect is outweighed by the danger of introducing such an irritant and volatile fluid. Not more than 2 ml. can be used at a time (2 ml. produces 400 ml. vapour). Bile salt solutions introduced in this way would need to be left in contact with a stone for a prolonged period. Clearly, this is not possible and one has to return to the idea that any efficient gallstone inhibitor or solvent will have to be a compound which is excreted in the bile by the liver.

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ANNUAL MEETING OF FELLOWS AND MEMBERS

Sheffield, 1961

THE NEXT ANNUAL MEETING of Fellows and Members will be held in Sheffield on Friday and Saturday, 24th and 25th November 1961.

The programme is to be as follows:

Friday, 24th November

Subscription Dinner for Fellows and Members at the Cutlers' Hall.

Saturday, 25th November

Morning	Scientific Exhibits and Films at the New Teaching Hospital.
Luncheon	in the Refectory of the University (given by the Board of Governors of the United Sheffield Hospitals).
Afternoon	The Annual Meeting of Fellows and Members in the Firth Hall of the University.
	Admission of Sir Howard Florey to the Honorary Fellowship of the College.
	The Arthur Hall Lecture by Sir Howard Florey (under the auspices of the Arthur Hall Trustees) in the Firth Hall.
Tea	at the Grand Hotel.

A further notice, giving fuller particulars, will be circulated to Fellows in September, together with an application form for attendance. Will Members of the College and other medical practitioners desirous of attending kindly write before 30th September to The Secretary, Royal College of Surgeons, Lincoln's Inn Fields, London, W.C.2.

ABNORMAL BLEEDING AFTER TOTAL BODY PERFUSION

Arris and Gale Lecture delivered at the Royal College of Surgeons of England
on
26th January 1961
by

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THE LAST DECADE has seen an increasing number of congenital and acquired cardiac defects corrected by open operations using an extracorporeal circulation. This method of surgical practice has presented many new and unique problems. One of these is a disturbance of the natural process of haemostasis which may lead to abnormal bleeding after total body perfusion.

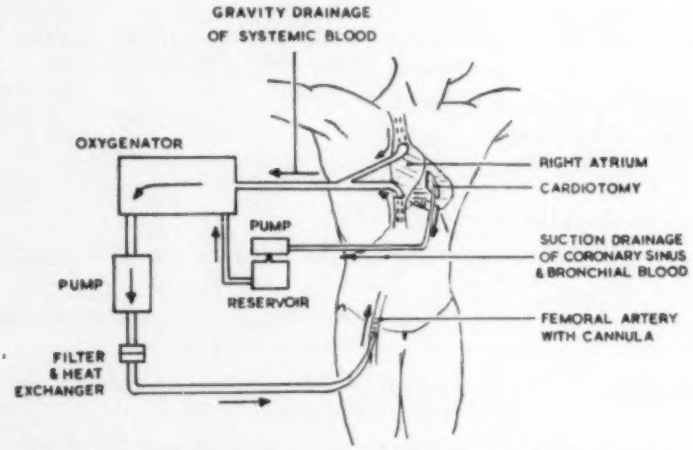

Fig. 1. Diagram of extracorporeal circulation. Arrows indicate direction of blood flow in circuit.

It is necessary during such procedures to divert blood from the heart and to maintain the cerebral circulation and the circulation to other vital organs of the patient while the heart and lungs are temporarily out of commission. The cardio-pulmonary by-pass must therefore perform for the patient the main functions of efficiently oxygenating the blood and pumping it back, at an adequate pressure, into the systemic circulation.

In a typical extracorporeal circulation (Fig. 1) systemic venous blood is drained, usually by gravity, from the venae cavae of the patient into the

external circuit through plastic cannulae. The blood is then passed over the surfaces of the oxygenator and from there is pumped through a filter and heat exchanger. The oxygenated blood is returned through a cannula to the patient as a retrograde perfusion of the femoral or iliac artery. Throughout the circuit, there are abrupt changes in tubing size and restricted orifices which give rise to turbulence of the blood in the by-pass. This, and the effect of the materials and surfaces in contact with the blood, can give rise to damage to the blood.

While the heart is open during the operation, the blood flowing from the coronary sinus and bronchial vascular system has to be removed from the site of operation in the heart by a mechanical suction system and passed through a settling reservoir into the oxygenator. Bubbling and frothing readily occurs in this suction system and contributes to further blood damage. The suction pressure is maintained at the minimum necessary to cope with the flow of blood, so reducing to a minimum the trauma to the blood.

In addition to these mechanical effects which influence the state of the blood after perfusion, the effect of the donor blood required to prime the external circuit and to replace any losses during operation must be considered. The volume of donor blood used during perfusion can be greater than the blood volume of the patient. This massive exchange transfusion will therefore influence the chemical and cellular nature of the patient's blood after by-pass. If the donor blood is poor in clotting factors, then dilution of those of the patient will occur as they mix during the time of perfusion. The amount of dilution will depend on the volume of donor blood used and its content of clotting factors, which is related to the duration of storage before perfusion and to the anticoagulant solution used. Heparin and E.D.T.A. (disodium salt of ethylene diamine tetra-acetic acid) are the solutions commonly employed, the latter requiring heparinization and calcification before being used in the pump-oxygenator circuit. Heparin was the anticoagulant of choice in the cases reported here and 25 mg. per 500 ml. of fresh blood was used.

It is also necessary to give the patient an anticoagulant in order to maintain the fluidity of his blood and to protect the platelets and plasma coagulation factors during the time of by-pass. Heparin was used for this purpose and was given to the patient immediately before perfusion in a dose of 2.5 mg. per kilogram of body weight.

Thus the main factors producing the cellular and clotting changes during total body perfusion are the trauma inflicted on the blood during its circulation, the effects of foreign contact surfaces and the exchange transfusion of donor blood. These may lead to a failure of haemostasis and produce abnormal bleeding after perfusion.

Another factor to be considered is heparin. Its presence is essential during perfusion to prevent clotting in the external circuit and it is equally

essential for it to be neutralized afterwards to restore normal coagulation in the patient. Its incomplete neutralization may also lead to post-perfusion bleeding.

Haemostasis

Normal spontaneous haemostasis and clotting are emergency functions of the body preventing loss of blood, and depend on the orderly correlation of vaso-constriction, the formation of platelet plugs and coagulation by the plasma factors (Fig. 2). Immediately on injury of a vessel the local stimulus causes vascular contraction and the endothelial surfaces tend to adhere and retract. Platelets, derived from the blood streaming out of the

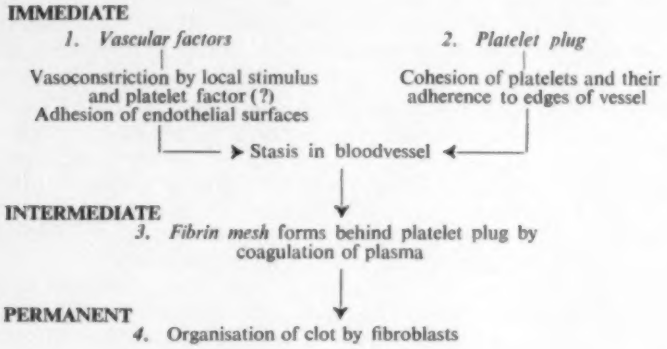

Fig. 2. Stages of natural haemostasis.

open vessel, adhere to the torn wall and to each other. The growing aggregation of platelets, some of which are swept away by the blood flow, gradually covers the cut end of the vessel to form a plug in two to three minutes. This plug produces stasis of the blood in the vessel behind it and initiates plasma coagulation. A fibrin meshwork then forms, giving firmness to the haemostatic plug and preventing renewed bleeding as the vascular contraction relaxes. Permanent arrest is produced by organization of the clot.

Coagulation

This is the final stage of haemostasis and is a complex series of actions which finally results in a fibrin clot. The modern theory of coagulation (Fig. 3) considers it as occurring in three stages. The first stage involves the generation of blood and tissue thromboplastin and can be considered as two processes, initiated in different ways but producing a similar end-product—thromboplastin. Contact with foreign surfaces causes platelet

and many plasma factors to react to form blood thromboplastin, and tissue factors released by tissue damage react with other plasma factors to form tissue thromboplastin.

The second stage entails the conversion of prothrombin to thrombin by blood and tissue thromboplastin.

The third and final stage is the enzymatic action of thrombin on fibrinogen to form a clot consisting of a network of branching strands of fibrin. It must be emphasized that coagulation is the property of platelets and plasma only, so that in tests involving whole blood the presence of cells tends to mask the end-point. Such tests give rise to many inaccuracies and have not been used in this investigation.

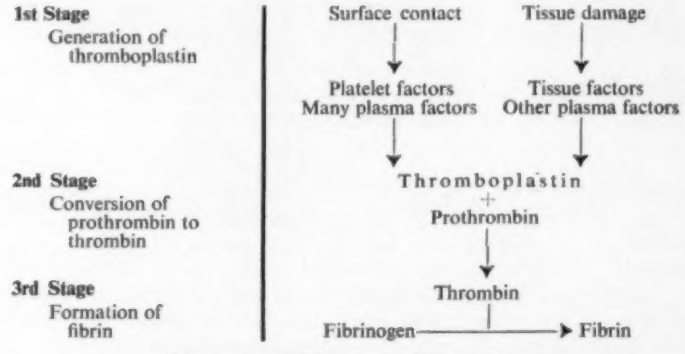

Fig. 3. Three-stage theory of coagulation.

Coagulation is normally in a dynamic state of equilibrium in the body (Laki, 1953; Biggs and Macfarlane, 1957; Allen, 1958) so that a balance is struck between insignificant intravascular coagulation on the one hand and the dissolution of clotting factors and products on the other. This latter process will receive further consideration when fibrinolysis is discussed below.

A deficiency of one or other of the various factors involved in natural haemostasis and coagulation can lead to a bleeding state. Such defects have been reported after the use of an extracorporeal circulation. A failure of vasoconstriction (Brown and Smith, 1958), a reduction of platelets and certain plasma factors (Gollub *et al.*, 1959; Perkins *et al.*, 1959) and fibrinogen depletion (Osborn *et al.*, 1955; Allen, 1958; von Kaulla and Swan, 1958) have been suggested as possible causes of abnormal bleeding after perfusion.

It is also possible for unneutralized heparin to lead to a failure of haemostasis after perfusion since this drug inhibits all the stages of blood coagulation.

The use of extracorporeal circulations is now a well-established procedure, but it still can be a hazardous operation. It thus behoves us to try to eliminate one of the complications of these open cardiac procedures by anticipating and determining as quickly and accurately as possible the cause of any abnormal bleeding, so that appropriate steps can be taken early.

In practice we have concentrated on three factors which may upset natural haemostasis after perfusion. The three factors are:

1. Unneutralized heparin.
2. Platelet reduction.
3. Fibrinogen depletion.

These defects have been studied because they can be rapidly detected and because corrective treatment is readily available should it be required.

Unneutralized Heparin

Previous studies to determine the amount of heparin neutralizer needed after perfusion have used coagulation tests which are poor indicators of heparin activity and are open to inaccuracies. It has been shown that the thrombin clotting-time fulfils the requirements of being simple and rapid to perform and is sensitive to low concentrations of heparin (Rothnie and Kinmonth, 1960a). It is performed by adding a thrombin-calcium solution to the plasma of the patient and timing the clot formation. The test is made a suitably sensitive indicator of heparin activity by adjusting the thrombin concentration to give an appropriate control clotting-time of 10 to 12 seconds. The test represents the final stage in coagulation and can therefore be influenced by the fibrinogen concentration in addition to heparin. However, the fibrinogen concentration must be reduced to 100 mg. per cent. or less before there is an appreciable prolongation of the thrombin clotting-time. This reduction is greater than that usually found after an average perfusion, so that any increase that may be found in the thrombin clotting-time after the administration of the antiheparin agent is most likely due to the presence of residual heparin.

In practice the test is applied in the following way. The control thrombin clotting-time is determined, using the patient's plasma taken before perfusion, and this is compared with the thrombin clotting-time of the plasma taken after the administration of the heparin neutralizer post-perfusion. If the time is similar to the control time, then the heparin has been completely neutralized. If greater, then additional doses of neutralizer are given until it is similar to the control pre-perfusion clotting-time. It is more rapid and convenient to give doses of heparin antidote based on the body-weight of the patient and to test for complete heparin neutralization in the way described than to assay the amount of heparin present in the patient after perfusion, and from this estimate the required dose of heparin neutralizer.

In our early experimental and clinical cases abnormal bleeding was a frequent occurrence and was found by this test to be due to unneutralized heparin. By increasing the dose of protamine and ensuring that there was no residual heparin, the incidence of oozing after perfusion was reduced and the immediate mortality from this cause was eliminated. There was, however, a considerable variation in the amount of protamine necessary for complete neutralization of the heparin. This inconsistency in the action of protamine led us at an early stage to the use of a new antiheparin agent—" polybrene " (hexadimethrine bromide—Abbott). The variable neutralizing activity of protamine appears to be due to competition for the agent, unlike polybrene, between the plasma proteins, especially fibrinogen, and heparin (Rothnie, 1960).

After an extensive trial of polybrene in experimental and clinical practice, a dose of 1.5 mg. of the agent for each milligram of the heparinizing dose given to the patient (polybrene-to-heparin ratio 1.5 : 1) was found to be consistently satisfactory in ensuring complete heparin neutralization (Rothnie and Kinmonth, 1960b).

Thus by adopting the routine described, the detection and elimination of residual heparin was found to be an important factor in reducing bleeding after perfusion as judged by improved experimental and clinical results.

Platelet reduction

Duplicate platelet counts were performed by the method of Brecher and Cronkite (1950) on the patient's blood before and immediately after perfusion. The difference between the means of these two counts represented the reduction due to perfusion. In a series of fifteen patients, who underwent open operation for the correction of varying cardiac defects, the mean reduction was 31 per cent. for an average perfusion time of 55 minutes. There was no direct relationship between the duration of perfusion and the percentage reduction of platelets. The platelet reduction was similar to that found by others and was not marked enough to affect normal haemostasis. This has not been found to be an important factor in the causation of bleeding after perfusion.

Fibrinogen depletion

This can occur in the body in two ways: (1) by fibrinolysis and (2) by the process of defibrination (Fig. 4). The fibrinolytic system is normally present in plasma and is a slow enzymatic process which is capable of increased activity under certain conditions. If this occurs, there is a rapid dissolution of fibrin, fibrinogen and other clotting factors. The exact mode of activation of this system is ill-understood, but stress and tissue and red cell damage have been implicated as activating factors after certain operations, including intra-thoracic procedures (Soulier *et al.*, 1952). The resulting destruction of fibrinogen can lead to defective haemostasis and

abnormal bleeding. Defibrination has been shown to be due to the release into the bloodstream of tissue thromboplastin which activates coagulation, producing small disseminated fibrin emboli (Schneider, 1950; 1956). The consequent consumption of clotting elements produces a depletion in the supply of plasma factors, especially fibrinogen, available for haemostasis, and this can also result in a bleeding state.

The main effect of the two processes of destruction, which may occur with perfusion, is a reduction in the circulating fibrinogen. It is not essential in practice to differentiate between them because, at present, the administration of human fibrinogen is the treatment for both processes.

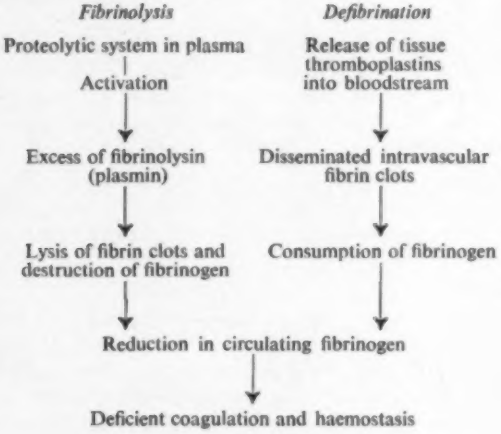

Fig. 4. Fibrinogen depletion.

The methods for detecting and estimating fibrinolytic activity and defibrination are many and varied and most are time consuming and laborious. The simplest methods are serial estimations of the fibrinogen concentration and observation of the incubated fibrin clot for dissolution. The concentration of fibrinogen in the plasma was estimated in this study by an absorptiometric method (Rothnie *et al.*, 1960). The principle of this estimation is to recalcify the citrated plasma and measure the opacity increase, due to the fibrin clot, in the absorptiometer. Assuming that all the fibrinogen is converted to fibrin, the opacity of the clot is related to the fibrinogen concentration in the plasma. The absorptiometer was calibrated with clot opacities containing known quantities of fibrinogen. These determinations were routinely carried out before and at intervals after perfusion since serial estimations are an index of the rate of destruction or consumption of fibrinogen which, if marked, may require fibrinogen replacement. The tubes containing the fibrin clots used for the fibrinogen

estimation were kept in a water bath at 37° C. for up to three hours. The rate of any breakdown of the clot within this time indicated in a crude way the degree of fibrinolytic activity.

In the series of fifteen patients already described, complete heparin neutralization was achieved by polybrene and no evidence of fibrinolysis was found. The fibrinogen concentration before and immediately after perfusion in each patient is shown in Figure 5. The mean reduction was 12 per cent. of the pre-perfusion level for an average perfusion time of 55 minutes. Most of the patients showed a reduction in fibrinogen, but two of them, initially low in fibrinogen, gained fibrinogen from the donor-

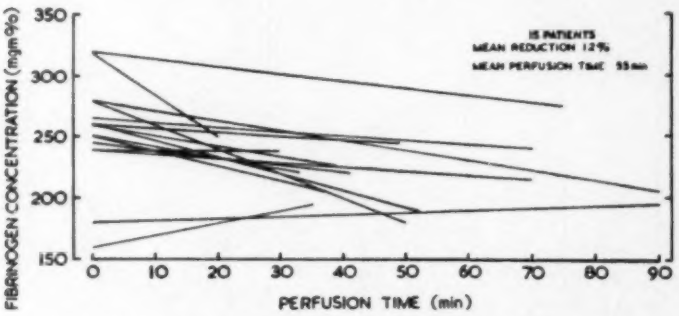

Fig. 5. The changes in fibrinogen concentration in fifteen clinical perfusions plotted to show the rate of change in individual cases.

blood during perfusion. There was no direct relationship between the degree of reduction and the duration of perfusion and it was therefore not possible to forecast accurately the changes that might occur due to perfusion. None of these patients had abnormal oozing after by-pass so that these changes in fibrinogen concentration were not marked enough to produce a failure of haemostasis.

Fibrinolysis has been detected in a number of patients after perfusion. One showed marked activity and fibrinogen depletion following a perfusion lasting two and a half hours. The others had mild fibrinolysis following perfusions of about one hour and there did not appear to be any specific differences between these and those not showing fibrinolysis. The duration of perfusion or age of the patient did not appear to bear any direct relationship to the incidence of fibrinolysis. The patient with marked fibrinolysis was a girl of thirteen years with a Fallot's tetralogy which was not improved by two previous operations. These made a third operation difficult and prolonged. The heparin was satisfactorily

neutralized with polybrene (1.5 : 1) and the prolongation in the thrombin clotting-time immediately after perfusion was found to be due to a reduced fibrinogen concentration—*ca.* 100 mg. per cent. (Fig. 6). The plasma clot showed rapid lysis within 20 minutes and a further specimen half an hour later produced scanty clot which was rapidly lysed. There was marked oozing from the various incisions. It was calculated that about 4 g. of fibrinogen was needed to replace this girl's deficiency and to give her some in reserve. An infusion of 4.4 g. of human fibrinogen was given

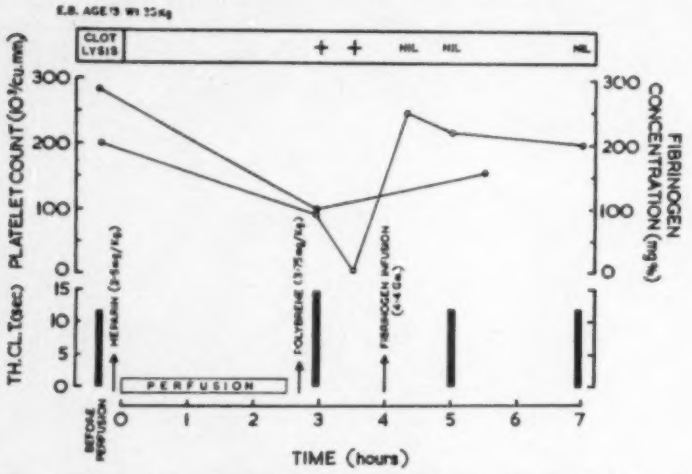

Fig. 6. Changes in the platelet count (●——●), fibrinogen concentration (o——o) and thrombin clotting-time (TH.CL.T.) after perfusion for 2½ hours. + indicates fibrinolysis. The effect of an infusion of fibrinogen is shown.

with a satisfactory return of the fibrinogen concentration to within the normal range and this was maintained. There was no evidence of any fibrinolytic activity after the infusion, and the thrombin clotting-time returned to that of the pre-operative control.

A typical example of a patient showing mild fibrinolysis (i.e. lysis of clot in one to two hours) is shown in Figure 7. This lady underwent perfusion for fifty minutes for the correction of mitral valve disease. The heparin was completely neutralized after perfusion by polybrene (1.5 : 1). The fibrinogen concentration before perfusion was 280 mg. per cent. and fell to 155 mg. per cent. about 30 minutes and 170 mg. per cent. about one and a half hours after perfusion. Both samples showed lysis in about one hour. No abnormal oozing was noted at the time of closure, but the immediate post-operative drainage was more than expected. Prior to a possible infusion of fibrinogen, a further estimation three hours after

perfusion showed a concentration of 240 mg. per cent. and no evidence of lysis. The infusion was not given because the fibrinolytic activity had ceased, the blood drainage had slowed and the fibrinogen concentration had returned to a normal value. The fibrinogen estimation two hours after this was 230 mg. per cent. with no clot lysis.

The other patients showing mild fibrinolysis and fibrinogen depletion also showed this cessation of activity within two hours from the end of

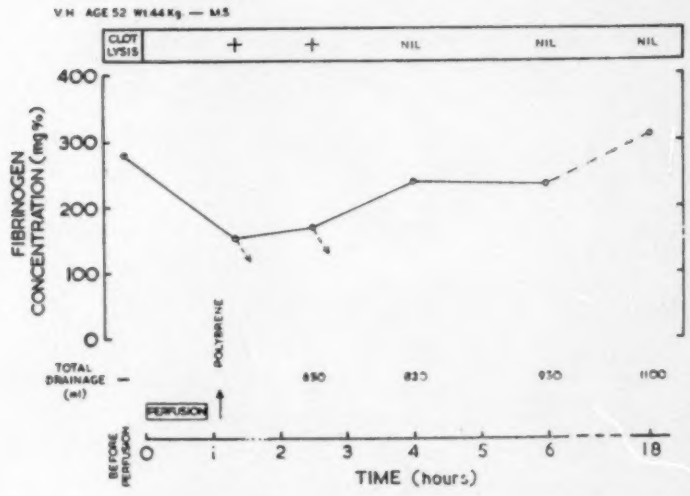

Fig. 7. Changes in fibrinogen concentration (o——o) after perfusion of 50 min. + indicates fibrinolysis. Post-operative blood drainage from the chest is shown.

perfusion followed by a gain in the fibrinogen concentration to within normal limits. None of these patients required fibrinogen replacement.

Fibrinolysis does appear to be more likely to develop and produce serious fibrinogen depletion as a result of a prolonged perfusion. Should it occur, it appears to be short-lived once perfusion has ceased and responds to the infusion of human fibrinogen. If the fibrinolytic activity is not marked the patient is able to replenish rapidly the circulating fibrinogen and so restore spontaneous haemostasis. There is at present no satisfactory method of reducing fibrinolytic activity and treatment consists of replacing the fibrinogen until its destruction ceases and a normal fibrinogen concentration is obtained.

Conclusions

The disturbance of coagulation factors resulting from the use of extracorporeal circulations sometimes leads to abnormal bleeding after perfusion. The incomplete neutralization of heparin at the end of perfusion was found to be an important factor in causation. It can be satisfactorily eliminated by the new antiheparin agent polybrene and should now cease to be a problem after perfusion.

Platelet reduction was not found to be of importance in causing abnormal bleeding.

With the elimination of residual heparin as a cause of bleeding, fibrinogen depletion was found to be an important cause of abnormal oozing after perfusion. It can be readily remedied by an infusion of human fibrinogen.

I wish to acknowledge my indebtedness to Professor J. B. Kinmonth, under whose direction this work was carried out, and to other members of the surgical perfusion team at St. Thomas's Hospital, London. This work formed part of a thesis submitted for the M.S. degree of the University of London.

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ANATOMICAL MUSEUM

THE MUSEUM WILL be closed throughout August.

THE ARCHITECTURAL HISTORY OF THE HUNTERIAN MUSEUM

by

Jessie Dobson, B.A., M.Sc.

Curator of the Anatomical Museum

IN 1783 THE lease of John Hunter's house in Jermyn Street, formerly the residence of his brother William, expired and by this date his collection of specimens was of great size and required better accommodation than was provided in their present situation. This he found in Leicester Fields, where No. 28 on the east side, the ground behind it, and the corresponding

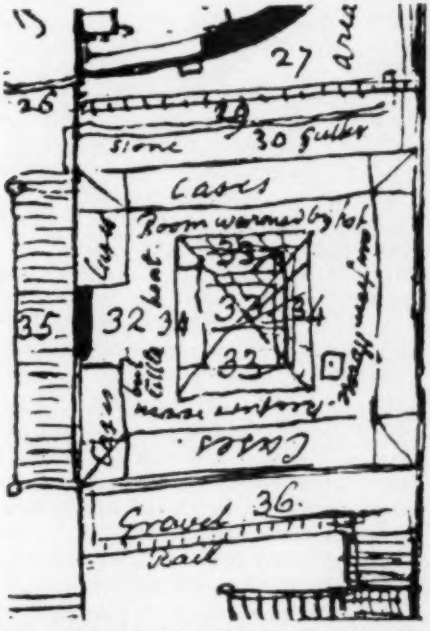

Fig. 1. Part of a sketch prepared by William Clift of John Hunter's house and premises in Leicester Square, showing the Museum section.

house facing Castle Street were available. Though the lease of these premises had only twenty-four years to run, he decided to purchase and immediately, on the ground between the two houses, proceeded to erect a lecture theatre and conversazione room and, above them, a museum, 52 ft. by 28 ft., lighted from the top, with a gallery all round (Fig. 1). The new buildings were not completed until 1785 and it is estimated that

the lease together with the alterations and additions to the original structure cost him no less than £6,000. Although Hunter and his family moved into Leicester Fields in the spring of 1783 and his lectures took place there from that time, the collection remained in Jermyn Street for another two years. A further alteration was required in 1792, when Sir Joseph Banks decided to give up his natural history collection, bestowing half on the British Museum, then in Montagu House, Bloomsbury, and giving the rest to John Hunter. William Clift, Hunter's new assistant, successor to William Bell, gives the information that a partition had to be moved between the front and back room of the first floor and Hunter's workroom moved from the first floor to the second, at considerable expense in order to provide room for the additional specimens.

After Hunter's death on 16th October 1793, although the house facing Leicester Fields was at once sold, that in Castle Street was retained for the accommodation of Clift, the housekeeper and the dissecting room attendant; and the collection remained in the museum behind it until 1806. The rent for these premises amounted to £150 a year, paid for the first half of the period out of Hunter's estate and for the rest by the College of Surgeons. By this date, the negotiations for the purchase of the collection by the Government and the arrangements for its custody in perpetuity by the Royal College of Surgeons had been completed. Furthermore, plans had already been drawn up for the building of suitable accommodation in Lincoln's Inn Fields, not only for the headquarters of the new College, but also for the housing and display of the 13,682 preparations left by Hunter, together with the cases and other things belonging to them or used therewith. The diversity of the specimens presented some difficulties for they ranged from the smallest items to massive articulated skeletons.

As early as 1800, meetings were held to discuss the design of the new building and on 2nd October of that year the committee set up for that purpose reported to the Court of Assistants that they "judged it proper fully to enquire into what appeared expedient to be done; and afterwards to balance the Competency of the Funds and other means of the College, to accomplish the Undertaking; concerning, that it would be better to adopt and by degrees perfect, a great and useful plan that would at all times prove suitable to the purpose and dignity of the College, than fix upon a Design, deficient either in Utility or Elegance because it would at once be executed". The Committee further recommended that building should be started in the following April; but in fact it was delayed for another six years.

At first it was contemplated that the site of the first house purchased by the Company in Lincoln's Inn Fields, No. 41 on the south side, would be adequate for the requirements of the College; but it soon became apparent that more ground was necessary to erect a building suitable for the housing of the museum, to provide accommodation for the Secretary of the College and for the Conservator of the Museum, and also for a

theatre " capable of containing not less than 200 persons ". Fortunately, while the preliminary plans were being discussed, No. 42 Lincoln's Inn Fields came into the market and was bought for £4,100 (Fig. 2). This meant, of course, that new plans had to be prepared and the estimated cost was correspondingly greater. Indeed, the mere shell of the building now designed by George Dance and James Lewis, the architects invited to undertake the work, was to cost £8,300.

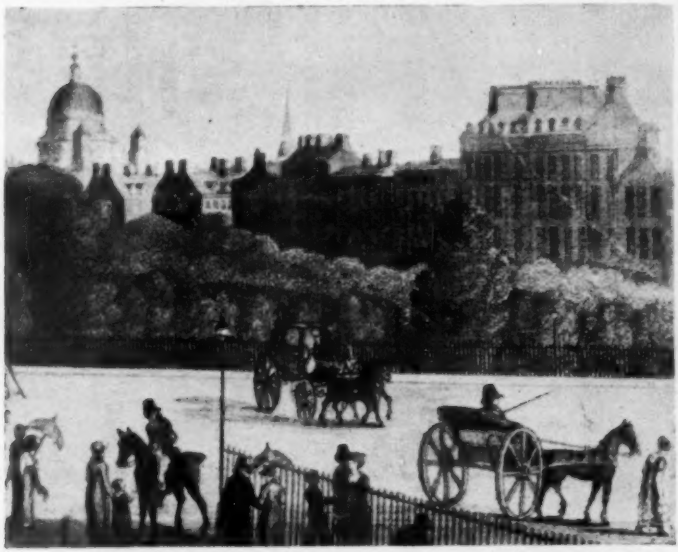

Fig. 2. Lincoln's Inn Fields *c.* 1800. The two tall houses on the far side of the square were demolished to provide the site for the first College building completed in 1813.

In 1806, therefore, when it was apparent that funds were not available for the undertaking and when, moreover, the matter of accommodation for the museum was becoming urgent since the Castle Street premises were in need of constant repair and in any case were now not even suitable for the storage and still less for the display of the specimens, it was decided that an " application should be made to Parliament for Aid towards the intended Buildings ". The amount deemed requisite was £15,000 and on 4th July 1806 the Chancellor of the Exchequer, in the Committee of Supply, moved that this sum " be granted to His Majesty for enabling the Royal College of Surgeons in London to erect a proper and commodious building for preserving and extending the Collection of the late Mr. Hunter, and a Theatre for the delivery of publick Lectures on Anato v

and Surgery and that the said Sum be issued and paid without any Fee or other deduction whatsoever ". As a token of appreciation of the valuable services rendered by Lord Grenville, first Lord of the Treasury, in these negotiations, it was resolved that a portrait of him should be commissioned; which painting was completed four years later and now hangs in the Secretary's room.

The building now proceeded with, apparently, no more than the normal delays and difficulty; but in 1810, it was found with dismay that the amount of money available was considerably less than would allow the building to be completed according to plan. A further petition was made to Parliament and the sum of £10,000 mentioned as being adequate. Doubtless to the surprise of the petitioners, the Committee of the House of Commons to which it was referred not only gave it favourable consideration but proposed to make an even larger grant—of £12,500, in fact—on condition that " an appropriate front should be added to the building ". In the end the total cost reached £46,770 15s. 11¾d., of which the Government had provided £27,500. Some little confusion now arose over the remuneration of the architects. George Dance and James Lewis were not partners. Though they had worked together in designing the plans for the College, they usually operated quite independently. On being asked to submit their account, they replied: " We have always charged and received the usual and established commission of 5 per cent. upon the expenditure of every Building which we have individually designed and of which we have directed and superintended the execution "; but they very courteously added that they would be perfectly satisfied with whatever sum was granted—which was, in fact, £2,338 10s., just 5 per cent. of the total expenditure, and £1,169 5s. each for more than eight years' work.

The building was finally completed and officially declared open on 13th May 1813, and the museum was ready for visitors—on Tuesdays and Thursdays only—from this date. The reason for making this arrangement was that as yet there were no printed catalogues for the collection and for fifteen years the Conservator, William Clift, was obliged to conduct parties of visitors round the museum to demonstrate and explain the contents, unaided except that at times members of the Board of Curators would undertake this duty when the visitors were very distinguished. This new venture on the part of the College proved to be quite successful. On 2nd July 1813, the Board of Curators were able to report to the Court of Assistants that though " they felt not a little solicitude concerning the success of their Provisions for opening the Museum, as they could not have any help from experience, upon the first Occasion, and as some of the proposed Regulations were necessarily adapted to temporary Circumstances, they have, therefore, greater pleasure in assuring the Court, that the utmost order and decorum prevailed on each Day of Inspection; and that the Visitors expressed themselves highly sensible of the dignity and importance of the Collection ". A contemporary description of the new

College states that " on entering the building the Great Saloon or Museum first engages the attention "—as well it might, for it was 91 ft. long and 39 ft. wide, with a gallery all round, lighted from the roof with circular " lanterns ". As many as possible of Hunter's own showcases had been used for the display of the preparations, but many others were specially designed and constructed (Fig. 3).

Fig. 3. A sketch by William Clift of the first museum of the College, designed by George Dance and James Lewis, opened on 13th May, 1813.

It seems unfortunate that this edifice that had taken so long in the building, had cost so much money and involved two petitions to Parliament, should have had such a short life; but, only 15 years after it was completed, the Conservator found that, as a result of the extensive additions that had been made to Hunter's original 13,682 specimens,

" great inconvenience arises from want of room "; and five years later the position was acute since only about half of the specimens could be displayed (Fig. 4). A building committee was appointed and held its first meeting on 23rd October 1833. The same difficulties had arisen in other parts of the College as in the museum and it was decided that an entirely new building should be designed. The museum was closed

Fig. 4. A sketch by Richard Owen, probably drawn about 1830, showing the overcrowding that necessitated building extra museum accommodation.

on 9th April 1834 and the work of reconstruction began at once. Notices had been issued inviting the submission of plans and five well known architects accepted. The designs of Charles Barry were considered most suitable, though these were modified later by the inclusion of certain features from the plans sent by other competitors. Barry estimated that the whole of the reconstruction could be accomplished for £16,439; in this, however, he sadly miscalculated and when, at the end of the three years' building programme, he submitted a bill of costs amounting to over £45,000, he was compelled to do a deal of explaining.

Barry demolished practically the whole of Dance's building, leaving only the walls of the museum and the portico—the " appropriate front " demanded by Parliament in 1810. To maintain its central position, one of the pillars had to be moved from the west to the east end. In his reconstructed museum, Barry had a " second gallery, putting in a flat roof lighted obliquely from the ceiling and substituting small cast iron columns

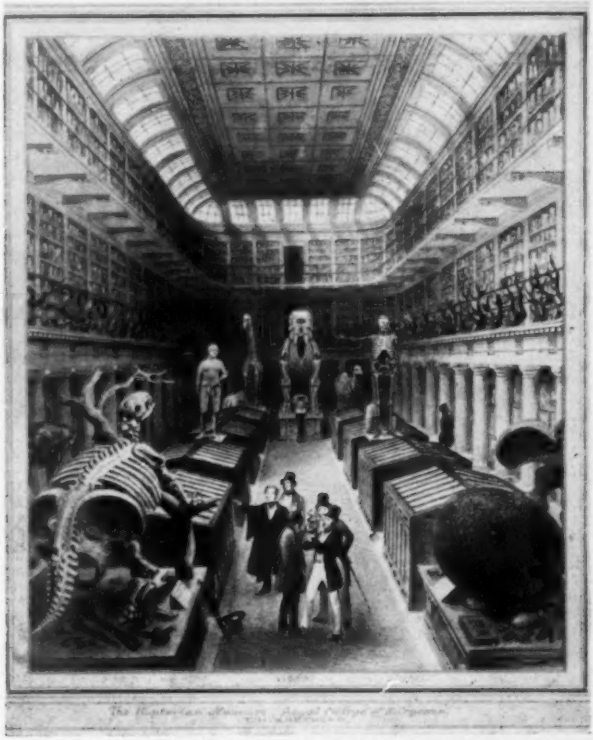

Fig. 5. Sketch by T. H. Shepherd of the larger of the two museums opened in 1837. This was later known as Room III. At the back of the room, in front of the Elephant, can be seen John Flaxman's bust of John Hunter.

for the present large wooden projections ". Leading from this room to the west was a smaller museum, on the site of the lecture theatre in the previous building, its design and fittings matching the larger museum in every way and communicating with it at ground floor and gallery levels. These rooms were now named the East Museum and the West Museum; and the new College building was officially opened on the anniversary of John Hunter's birth, 14th February 1837 (Fig. 5).

Ten years later the collection had more than doubled its size and Richard Owen, then Conservator, complained, like his predecessor, William Clift, of the shortage of space for proper display. This was not surprising, for Owen's zoological investigations resulted in the acquisition of such specimens as the Megatherium and the Glyptodon, and the recently founded Zoological Society of London provided abundant material for the expan-

Fig. 6. Part of the Middle Museum, looking into the East Museum, later Rooms IV and V.

sion of the original collections. At a special meeting of the President and Vice-Presidents held on 27th November 1850, the question of making an extension of the museum on land adjoining the existing buildings, purchased from Alderman Copeland in 1847, was discussed; and at a further meeting held on 8th January in the following year, Charles Barry submitted estimates for the construction of another museum room east of the existing smaller one, such room to conform with the general design of the other two museums. Barry's first plan was for a room 50 feet by 30 feet,

but this was judged not to be large enough to provide any material alleviation of the congestion in the museum. After more than a year of discussion, the plan finally approved was for a room 100 feet by 40 feet, with two galleries, the cost of which was estimated at more than £20,000. In addition, there were to be further alterations and additions to other parts of the building and a decision was reached in

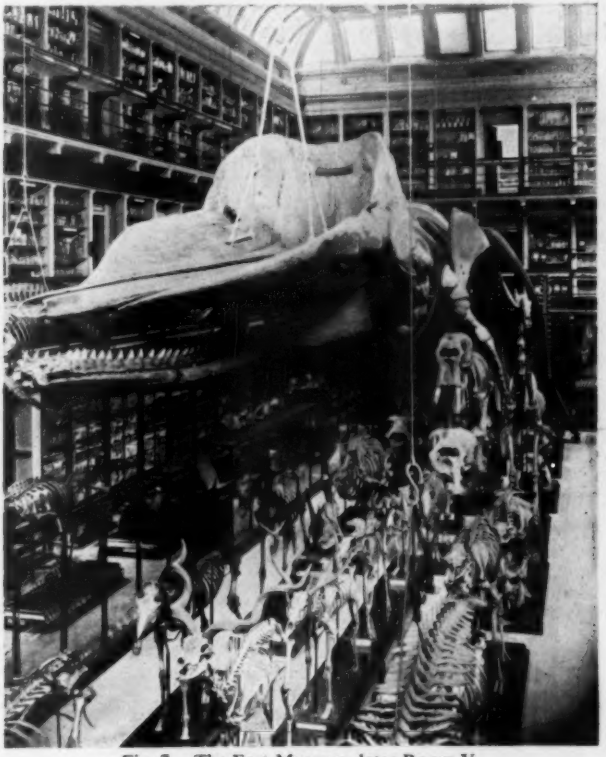

Fig. 7. The East Museum, later Room V.

Council on 9th January 1851 to petition Parliament for a grant to meet these expenses. It was reported on the following 24th July that £15,000 had been voted for this purpose. The work was begun at once and was completed in 1855, and once again it was possible to display all items in the collection in what came to be known as the East, Middle and West Museums (Fig. 6).

John Thomas Quekett succeeded Richard Owen as Conservator in 1856 and although during the next six years until his death in 1861 he added

almost 7,000 preparations to the museum, these caused no embarrassment as regards accommodation since they consisted of histological specimens. These formed a valuable series complementary to and explanatory of many of the items in the main collection. It was Quekett's successor, William Henry Flower, who created a further problem of space during the 23 years of his conservatorship, for his main concern and interest was to develop that section of the museum devoted to human and comparative osteology which, until this time, had been less well represented than the others.

Fig. 8. The new museums, Rooms I and II, completed in 1891. The design of the roof differs from that of the other rooms.

This object involved the preparation and display of complete articulated skeletons of all the vertebrate animals, together with a comparative series of individual bones and racial types. The problem arose not so much from the number as the size of the exhibits, which included whales, elephants, giraffes, camels, crocodiles and sharks. Some were slung from the roof and could be viewed with ease from the galleries (Fig. 7). The difficulties were manifold, yet it was never contemplated that the project of making the College Museum of Human and Comparative Anatomy, Osteology and Pathology the finest in the country should be abandoned.

In 1881 the Natural History Museum at South Kensington was completed and two years later William Henry Flower was appointed to the

post of Superintendent in succession to Richard Owen. The new Conservator of the Hunterian Museum was Charles Stewart, who was at once faced with the recurrent need for more space. Once again the College endured the discomforts of rebuilding and extension. The house numbered 43 Lincoln's Inn Fields, bought by the decision of the Council in 1860, was demolished and the site used for the building of two more museum rooms (Fig. 8).

Charles Barry had died in 1860 and the new architect was Stephen Salter, who was allowed to make only slight modifications in Barry's original museum design. The cost of the alterations and extensions, which were completed in 1891, was £19,000, a sum provided from the munificent legacy of more than £200,000 left to the College by Sir

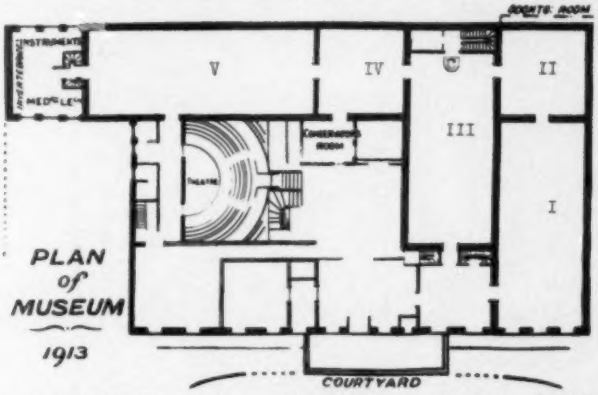

Fig. 9. Rooms III, IV and V were designed by Charles Barry; I and II by Stephen Salter.

Erasmus Wilson. The museum now consisted of five spacious intercommunicating rooms, each with two galleries; and there were several smaller side- and store-rooms (Fig. 9). The collection that had started in 1800 with the nucleus of John Hunter's 13,682 specimens had now increased to some 63,000 items, a comprehensive representative display of normal and morbid human and comparative structure.

The only thing that appears to have disturbed the serenity in the museum during the next 40 years was the introduction of artificial lighting. Up to this time, visitors had been admitted only during daylight, a regulation that drastically curtailed the activities of students especially during the winter months. It was so recently as 1917 that, on the request of the Museum Committee, the President authorized the expenditure of £47 17s. 6d. " for temporarily installing the electric light in Rooms I and II of the Museum for illuminating the exhibition of War Office specimens of military injuries on dark or foggy days ". Although this appears to have

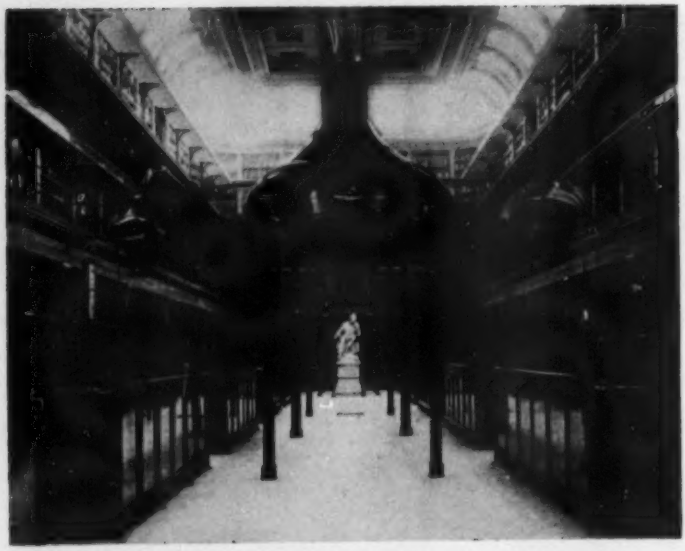

Fig. 10. Room III of the Museum before "the electric light" was installed.

Fig. 11. Room III after 1924.

met with everyone's approval, nearly seven years passed before permanent lighting was installed. It was authorized for Room I at a cost of £115 17s. in July; and in Rooms II and III, costing £288 9s. 6d., in December 1924. The other two rooms were similarly improved for the sum of £232 16s. (Figs. 10 and 11).

When it seemed that war was inevitable and imminent, as many as possible of the more valuable and, in particular, the Hunterian specimens were moved to what were considered to be bomb-proof basement premises; but obviously the bulk of the collection had to remain in the museum.

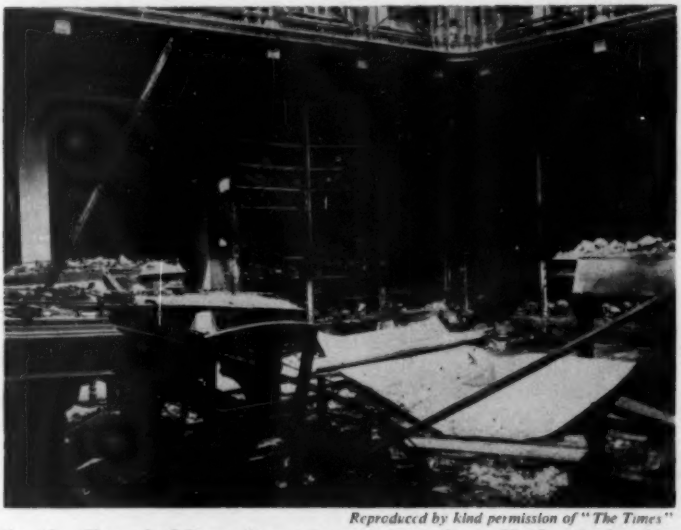

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Fig. 12. The end of Room I near the entrance to Room II after the bombing in 1941.

If the decision had been made then to move it away from London, the dismantling, packing and transport would have presented a major difficulty, even had the labour and time involved been considered justified at that time of national crisis. When the College was bombed on the night of 10th/11th May 1941, Rooms IV and V were destroyed, together with their contents, mainly the larger examples of comparative osteology. The other three rooms suffered less structural damage, but fittings, cases and specimens were affected to varying degrees by fire and water (Fig. 12). The remaining preparations were at once sent to various centres away from the London area; it was estimated that only about a third of the original number had survived.

The relics of the collection were brought back to the College in 1946 and accommodated in Rooms I, II and III, which has been repaired for use as museums and lecture rooms until such time as rebuilding could be undertaken. The year 1960 not only marked the centenary of the death

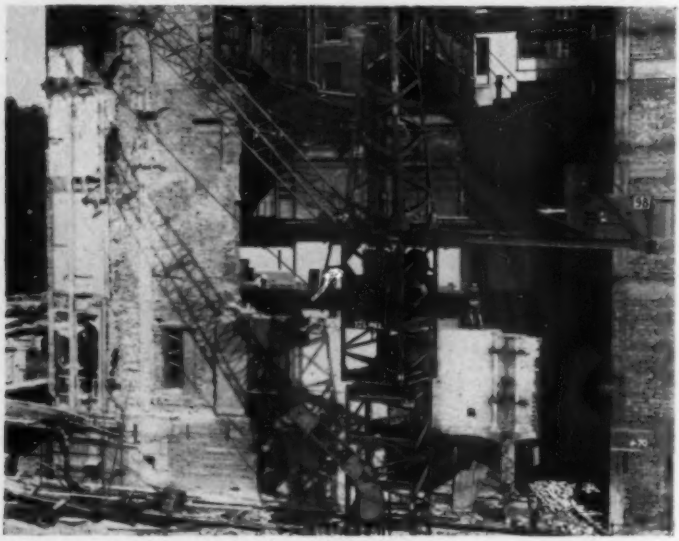


Fig. 13. The demolition of Room I; the last of the old museums.

of Charles Barry, it also saw the demolition of the last remnants of the museums he designed (Fig. 13). The new Hunterian Museum will, it is hoped, have its official opening on 13th May 1963, the 150th anniversary of the first such ceremony in the Royal College of Surgeons.

AWARD OF THE HONORARY FELLOWSHIP

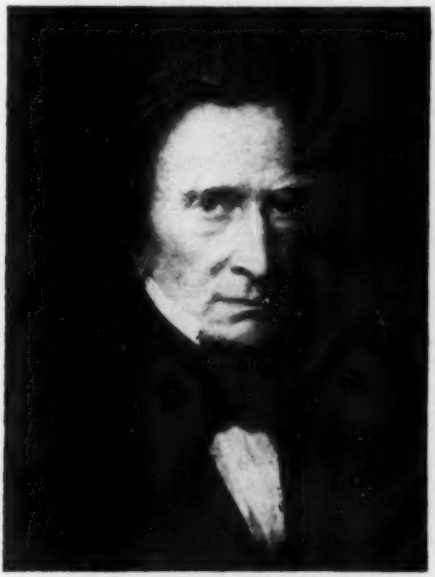
THE HONORARY FELLOWSHIP of the Royal College of Surgeons of England has been awarded to Sir Howard Florey, M.D., F.R.C.P., President of the Royal Society and Professor of Pathology in the University of Oxford, and Dr. Owen H. Wangensteen, M.D., Chairman, Department of Surgery, University of Minnesota, and former President of the American College of Surgeons.

PAST PRESIDENTS OF THE COLLEGE

(8) SIR BENJAMIN BRODIE, Bt.

President in 1844

SIR BENJAMIN BRODIE, the only man who has been President of the College and President of the Royal Society, was born the son of a country parson in the year that William Hunter died, 1783. His father's sister Elizabeth married Thomas Denman, Hunter's successor as the leading gynaecologist in London; several of the Brodies were prominent lawyers. Brodie was no doubt helped by his connection with this intellectual aristocracy, in which his Denman cousins also shone: the younger Thomas Denman



Sir Benjamin Brodie, Bt.

became Lord Chief Justice, while Sophia Denman married Matthew Baillie, nephew and heir of the Hunters and physician to the King.

Brodie's own ability and industry won him an outstanding position in surgery in the first half of the 19th century. When Sir Astley Cooper died in 1841, Brodie was undoubtedly the leading surgeon in England, and he became President of the College in 1844. He was reputed a severe man, devoted to duty and determined in his opinions, but he had many friends, many admiring pupils, and a happy family life. He married, in 1816, Anne Sellon, daughter of an eminent lawyer, a Serjeant-at-Law. The eldest of their three children became Professor of Chemistry at Oxford.

Brodie learned anatomy from James Wilson during 1801–02 in William Hunter's old school in Great Windmill Street, and then entered St. George's Hospital as a pupil of Everard Home, John Hunter's brother-in-law and former assistant; this was just 10 years after Hunter's death. Brodie became Home's personal surgical assistant, and worked with him on comparative anatomy at the Hunterian Museum. He staunchly defended Home's destruction of Hunter's papers, denying the well-founded allegation that Home set his own name to unpublished work by Hunter.

Home brought Brodie into the circle of the Royal Society, and he was elected F.R.S. in 1810. At the early age of 28 he was awarded the coveted Copley medal for his papers " The influence of the brain on the action of the heart " and " The effects produced by certain vegetable poisons—alcohol, tobacco, woorara ".

At the College Brodie was Hunterian Professor from 1819 to 1823, lecturing on anatomy, physiology, and surgery; he was elected to the Council in 1829, having been surgeon to St. George's since 1822. He was also surgeon to King George IV, and attended the King during his last illness in 1830. King William IV appointed him Serjeant Surgeon in 1834 and created him a baronet. He retired from St. George's in 1840, and devoted his energies to a successful reform of the College's constitution. As senior vice-president in 1843 he was instrumental in securing the Charter which instituted the Fellowship; he had himself planned this method of hall-marking the fully equipped surgeon, which was destined to advance the status and prestige of British surgery.

Brodie also took the lead in establishing the General Medical Council and was appointed its first President in 1858. In the same year he was elected President of the Royal Society.

Brodie wrote on many anatomical and surgical topics, and also on neurology and even psychology, for his interests ranged wide. He was a remarkably clear and persuasive teacher and lecturer. His best remembered work is recorded in his successful book on *Diseases of Joints*, published in 1818 and four times revised. By an irony of fate he died on 21st October 1862 from the effects of a malignant tumour arising on the site of an old dislocation of the shoulder. He was 79.

His great-grandson, the present Sir Benjamin Brodie, generously gave the College a few years ago the large bronze plaque designed by William Wyon, R.A., in 1841; it is a fine expression of Brodie's handsome profile. The marble bust commissioned by the Council from Henry Weekes, R.A., a few months before Brodie's death, and posthumously carved, is less attractive.

W. R. L.

PRESIDENT'S DINNER

AT THE BEGINNING of this year the Council had decided to institute an annual dinner, to be known as "The President's Dinner", designed to bring together the present and past Members of the Council and the present and past Members of the Court of Examiners, together with their ladies: in addition there were to be a few invited guests—Fellows who had distinguished themselves in surgery without ever belonging to either of the main statutory bodies of the College.

The first of these dinners was held in the Edward Lumley Hall on 24th May, and it was clear that it not only served its intended purpose well, but was greatly appreciated by those who were fortunate enough to be present.

The President (Sir Arthur Porritt) was in the Chair and he proposed the toast of "The Court and Council", pointing out that, whereas in past years there had been much cross-membership between the two bodies, this was now infrequent and there was need for the Members of Council and Court to meet together, being the two bodies most concerned in conducting the affairs of the College. He traced the origin of the Court of Examiners back to 1540, the Council of that date being known as the Court of Assistants and only obtaining its present title in 1822. Throughout its history the Court had maintained the standard of surgery, the real essence of the College, and for that great service the Council was extremely grateful.

To celebrate this evening's event the President promised to give a badge for the use of the Chairman of the Court of Examiners.

He then referred to the striking increase of activity and widened horizon of the College during the last 20 years and the advances still in progress: the most recent recognition of this work was the gift of £250,000 from the Wolfson Foundation, the promise of which had been received by the Council with enormous gratitude.

In welcoming the guests the President mentioned in particular those Fellows who, without having served on either Council or Court, had rendered great services to Surgery and therefore to the College. He paid tribute to the ladies and their unselfish support of surgeons and expressed his appreciation of the work of the administrative staff. He concluded that the dinner was a successful experiment which should be followed up.

Mr. Illtyd James, Chairman of the Court of Examiners, replied and first thanked the President for his promise of a Badge—the gift would be regarded as a great compliment and would be cherished and honoured as a token of the close relationship of Council and Court.

He also thanked the President for his praise of the Court, and recalled that at the first Fellowship Examination in 1844 the Court had consisted entirely of Members of Council but in general surgery it now had no representative of the Council at all. So he was very grateful for this function, rendered all the happier by the presence of the ladies. He paid tribute to the past members of the Court who had handed on its valuable traditions to their successors, and referred to the various specialties now represented.

He then recounted some examination anecdotes and enumerated light-heartedly the several Joneses now serving on the Court. Addressing those arrayed before him, he then concluded: " I have much pleasure in informing you that your performance has been entirely satisfactory to this Court. Your success will be reported to the Council of the College, with whom it rests to confer upon you, in a year's time, another dinner."

The following is a list of those present:

Council: Sir Arthur and Lady Porritt, *Sir Stanford and Miss Cade, *Professor Digby Chamberlain, *Sir James and Lady Paterson Ross, Sir Russell and Lady Brock, *Sir Eric and Lady Riches, Mr. and Mrs. C. Naunton Morgan, *Professor and Mrs. Charles Wells, *Professor and Mrs. R. Milnes Walker, *Mr. and Mrs. Harold Edwards, *Professor and Mrs. F. A. R. Stammers, Mr. and Mrs. J. C. Barrett, *Mr. and Mrs. F. W. Holdsworth, *Mr. and Mrs. R. V. Cooke, Mr. and Mrs. H. Osmond-Clarke, Professor and Mrs. L. N. Pyrah, Mr. and Mrs. C. Gill-Carey, Professor and Mrs. M. A. Rushton, Mr. and Mrs. T. Keith Lyle (also Court), Mr. and Mrs. H. G. E. Arthure, Dr. and Mrs. Geoffrey Organe, Professor and Mrs. D. W. Smithers.

Court of Examiners: Mr. and Mrs. Illtyd James, Mr. and Mrs. D. Ioan-Jones, Mr. and Mrs. Clive Butler, Mr. and Mrs. S. H. Wass, Mr. and Mrs. R. H. Franklin, Mr. and Mrs. Clifford Jones, Mr. and Mrs. David Trevor, Mr. and Mrs. E. G. Muir, Professor and Mrs. A. L. d'Abreu, Mr. and Mrs. W. M. Capper, Mr. and Mrs. Hugh Reid, Mr. and Mrs. A. H. M. Siddons, Professor and Mrs. D. M. Douglas, Mr. and Mrs. W. A. Mill, Mr. and Mrs. G. H. Macnab, Professor and Mrs. R. S. Pilcher, Mr. J. D. T. Jones, Mr. and Mrs. R. G. Macbeth, Mr. and Mrs. A. G. Cross, Mr. and Mrs. C. J. B. Murray, Mr. and Mrs. A. H. Hunt, Mr. and Mrs. C. A. Keogh.

Past Council: *Sir Henry Souttar, *Sir Max Page, *Sir Heneage and Lady Ogilvie, *Sir Cecil and Lady Wakeley, *Mr. and Mrs. Lionel Norbury, Sir Harry and Lady Platt, *Mr. and Mrs. J. B. Oldham, Dr. and Mrs. H. Guy Dain, *Sir Victor and Lady Negus, Sir Brian and Lady Windeyer, *Mr. J. H. Doggart, Dr. and Mrs. O. C. Carter, *Mr. Myles Formby, Dr. and Mrs. E. Rohan Williams, Mr. Frankis and Miss Evans, Mr. and Mrs. A. C. H. Bell, Sir Wilfred and Lady Fish.

Past Court: Sir Claude and Lady Frankau, Mr. and Mrs. P. J. Moir, Mr. and Mrs. J. B. Hume, Mr. W. H. C. Romanis and Mrs. Brady, Mr. and Mrs. V. C. Pennell, Mr. and Mrs. John Gardham, Sir Stewart and Lady Duke-Elder, Mr. and Mrs. F. W. Law, Professor and Mrs. V. W. Dix, Mr. A. Mekie Reid, Sir Ralph and Lady Marnham, Mr. and Mrs. A. W. Kendall, Mr. and Mrs. E. L. Farquharson, Mr. and Mrs. G. Qvist, Mr. and Mrs. J. H. Cobb, Mr. and Mrs. C. W. Flemming, Mr. and Mrs. E. S. Lee.

Invited Guests: Professor and Mrs. T. Pomfret Kilner, Sir Denis and Lady Browne, Mr. and Mrs. H. P. Winsbury-White, Mr. and Mrs. Vincent Patrick, Mr. George Armitage and Mrs. Ropner, Mr. and Mrs. C. E. Kindersley, Mr. and Mrs. H. Hamilton Stewart, Mr. and Mrs. S. A. S. Malkin.

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* Also Past Court.

ADMISSION TO THE COURT OF PATRONS

On 29th June the President and Council gave a luncheon in honour of Mr. Isaac Wolfson on the occasion of his admission to the Court of Patrons. Also present were Mrs. Wolfson, Mr. Leonard Wolfson and Sir John Cockcroft, all Trustees of the Wolfson Foundation, and Sir Harold Redman, Director and Secretary of the Foundation.

Left to right: Sir Arthur Porritt (President), Mrs. Wolfson, Mr. Isaac Wolfson, Lady Porritt, Professor Digby Chamberlain (Vice-President), Sir John Cockcroft and Mr. Leonard Wolfson.

At the brief ceremony of admission, which took place before the luncheon, the President, Sir Arthur Porritt, spoke thus:

" Ladies and Gentlemen: As you know, this is a quite informal occasion, but nevertheless I just want to perform a very simple formal ceremony to admit Mr. Isaac Wolfson as a Member of the Court of Patrons. Before I do so, I would like most heartily to welcome Mr. Isaac Wolfson and the Trustees of the Wolfson Foundation, Mrs. Isaac Wolfson, Mr. Leonard Wolfson and Sir John Cockcroft. I am quite sure Mr. Isaac Wolfson would be the first to say that the help of his wife, son and fellow Trustees is invaluable to him in the magnificent work his Foundation does. It is a very great regret to us that the other Trustees have been unable to come for various reasons; Sir Stanford Cade and Lord Evans are both examining, Lord Nathan, Lord Birkett and Professor Goodhart have also written, and sent good wishes. However, it is a great delight to us to see you, Sir, and your family with us, and I would like formally to tell you that at the last meeting of the Council you were unanimously and with acclamation elected a Member of the Court of Patrons of the College. In honouring you we are honouring ourselves, and I shall content myself at the moment with saying that we are enormously grateful to you. And so by the authority of the Council and by virtue of my office of President of this Royal College, I hereby admit you to the Court of Patrons."

After the Badge of the Court of Patrons had been placed round his neck by the President, Mr. Wolfson replied in the following words:

" This is indeed a great honour for me. When I look back to forty years ago when I first came to London, even in my wildest dreams I would never have believed that I should receive this honour, and I can assure you that I am indeed very grateful. Anything our family does for the Royal College of Surgeons is a pleasure, as we greatly admire all the work you are doing."

When proposing the health of Mr. Isaac Wolfson after lunch was over, the President spoke as follows:

" What I have to say is something I say from the bottom of my heart. Now that you are one of us, Sir, I want to be able to thank you again most wholeheartedly and most genuinely because this gift of yours to us has made a vast difference to our lives in this College. I am quite sure that you must get tired of superlatives to describe your gifts. I myself have never been in the happy position of giving away a quarter or half a million pounds, but it must be a little embarrassing to have such fulsome thanks poured on you, although in this case I trust a heart-warming operation, because they are very much meant. As half-a-Scot myself, I can say to you how especially we appreciate this gift, because it is the first big gift this College has had from Scotland! We realize that the acceptance of your gift is the acceptance of very great responsibility and we can assure you that we shall do everything within our power to fulfil this responsibility.

" We as a College were delighted with the wording that came with your gift; the fact that you showed an appreciation of the work of this College, both in this country and elsewhere, was something that meant a great deal to all the Members of Council; and I may say that your desire to attach your gift to the Hunterian Museum, the very heart and tradition of this College, was something which enhanced its value enormously. We realize that this is a special gesture on the part of the Foundation—that in making a donation of this magnitude to an ideal and not to bricks and mortar is something which is a great compliment to this College and it is also a great impetus and incentive. If one is dealing with entirely material things, that is one thing; but you have by your gift given us a real stimulus. We hope as we get to know you better as a member of our Court of Patrons (who, I may say, are not only appointed for what they give but because we hope and expect and pray that their friendship and interest will be continued in the object for which they originally gave their donations), we shall be able to look forward to seeing you here on many occasions so that you may get to know this College and in so doing know your gift was worth while.

" We have here a vision. We have in the past been accused of following a policy in recent years which was a little euphoric and venturesome, but I think we are quite happy to accept both these terms. Our policy is virile, dynamic and entirely in keeping with the times in which we live. Your gift has done an enormous amount of good because we feel it was given to fulfil these ideals—difficult ideals, but something well worth doing.

" When you get to know us well you will realize that a great many things are happening here. Our vision is founded on the art and science of surgery, but we would like to suggest that the work done in this College has a much wider field; we are concerned also with education and with scientific research on quite a wide front, and so, we hope, with the betterment and happiness of mankind, not only in this country, but also in the Commonwealth and the rest of the world. I trust, therefore, that you and your Trustees will feel that your gift was not only specifically given to surgery but given with a wider view to include science and the humanities. I hope it may seem to you, as it certainly does to us, a most invaluable gift."

The President then went on to refer to the Court of Examiners, which provided a guarantee to assure people of the highest standards of surgery; to the eighty resident postgraduate students from all over the Commonwealth and various other countries, who were not only being taught British surgery, but were also being imbued with the British way of life; the many lectures and courses in surgery and basic medical sciences; the

two Faculties, both very much alive, of Anaesthetists and of Dental Surgery; the Joint Secretariat, which was responsible for the co-ordination of all other specialties in surgery; the two flourishing journals being run under the auspices of the College; the social life of the College which was so important in producing a happy and cooperative atmosphere in the College; and to the magnificent museum and the library. He continued:

" These are just some of the activities which flourish at the College which we are so proud you have joined to-day, and we hope that the interest that you and your Trustees have shown in us as well as being an enormous stimulus to us will be a pleasure to yourself. You have backed an ideal and it will be our aim and intention to make that ideal worth while. Our thoughts are just these: you have not only opened the doors of the new Hunterian Museum, but also of a vista of ever-increasingly valuable work for the ultimate good of mankind as time goes on."

Mr. Wolfson, in his reply, referred to the enormous profits which accrued from the Great Universal Stores each year, which enabled his Trustees to make these philanthropic benefactions. He then subdivided the total of these profits to cover weeks, days, hours and seconds, with a facility and speed which were the envy of some of his less mathematically minded hearers. He then went on to say:

" The next thing I would like to mention is that when talking to Lady Porritt before lunch she mentioned Lord Webb-Johnson. He lived below me for a number of years and on many occasions he said to me, ' You must come round to the Royal College of Surgeons '. This was about ten or twelve years ago, and I wish I had taken his advice before this. I must say that Sir Arthur is a high-powered salesman, as Lord Webb-Johnson would have let me off with far less than Sir Arthur persuaded me to give! Now I think John (*Sir John Cockcroft*) would like to say a few words on behalf of the Trustees. But before he does so, I would wish to express our gratitude for your hospitality to-day and say how appreciative we are of the kind words you have said about us. And let me assure you that we know we will get great benefit from what you are doing for us all here in the College."

Sir John Cockcroft then spoke very briefly and said that as the sole scientific member of the Trustees present, he would like to assure the College that they attached great importance to the scientific work being done at the Royal College of Surgeons, and hoped to hear more about it on future occasions.

APPOINTMENT OF FELLOWS AND MEMBERS TO CONSULTANT POSTS

A. R. HARRISON, M.R.C.S.	Part-time Consultant Physician, St. Peter's Hospital for Stone.
L. P. THOMAS, F.R.C.S.	Consultant Surgeon, Royal Gwent Hospital, Newport.

The Editor is always glad to receive details of new appointments obtained by Fellows and Members, either through the Hospital Boards or direct.

BOOKS ADDED TO THE LIBRARY

April–June 1961

(Books without date are new publications)

Anaesthesia

ADRIANI. Pharmacology of anaesthetic drugs. 4th edition.
MEDUNA. Carbon dioxide therapy. 2nd edition. 1958.

Anatomy

BLOOMER AND OTHERS. Surgical anatomy of the broncho-vascular system. 1960.
NICOLESCO. Atlas of morphology of visceral nerve endings. 1958. Gift of Medico-Pharmaceutical Institute, Tirgu-Mures, Rumania.
See also under Cytology.

Biochemistry

CHARGAFF AND DAVIDSON. Nucleic acids, vol. 3. 1960. Continuation of series: vols. 1–2 published 1955.

Breast

INGLEBY AND GERSHORN-COHEN. Comparative anatomy, pathology and roentgenology of the breast.

Cancer

COLEY. Neoplasms of bone. Gift of Messrs. Hoeber, publishers.
MUIR. Carcinoma of the colon.

Cardiology

HOLLAND KLEIN. Chemistry of heart failure. 1960.
MERENDINO (editor). Prosthetic valves for cardiac surgery.
MORGAN-JONES (editor). Modern trends in cardiology. 1960.

Chemotherapy

FLOREY. Clinical application of antibiotics, vol. 2. Completion of series: 1, 1952; 3, 1957; 4, 1960.

Cytology and Microscopy

BOYD AND OTHERS. The electron microscope in anatomy.
POLICARD ET BAUD. Les structures inframicroscopiques normales et pathologiques des cellules et des tissus. 1958.

Dental Surgery

HOLLINSHEAD. The survey of dentistry in the U.S.—final report. Gift of Prof. B. Cohen.

Endocrinology

GARDINER-HILL. Modern Trends in endocrinology, 2nd series.
See also Thyroid.

Haematology

MACFARLANE AND ROBB-SMITH. Functions of the blood.
PICKERING AND OTHERS. The treatment of hypertension.
WALKER (editor). Thrombosis and anticoagulant therapy. Gift of Prof. R. B. Hunter.

Neurology

ADRIAN. Factors in mental evolution (Romanes lecture). 1960.
COOPER. Parkinsonism, its medical and surgical treatment.
MCCULLOCH (editor). Human decisions in complex systems. (New York Academy of Science.) Gift of Sir Cecil Wakeley, Bt.
GARLAND (editor). Scientific aspects of neurology.

Neurosurgery

MACCARTY. Surgical treatment of intracranial meningiomas.
POTTER. Practical management of head injuries.

Ophthalmology

DUKE-ELDER. Anatomy of the visual system. (System of Ophthalmology, vol. 2.)
SCHEPENS (editor). Importance of the vitreous body in retina surgery.
SMELSER. The structure of the eye.
WEALE. The eye and its function.
WOLFF. Anatomy of the eye and orbit, 5th edition by R. J. Last.

BOOKS ADDED TO THE LIBRARY

Orthopaedics

BARNETT AND OTHERS. Synovial joints, their structure and mechanics. Gift of Messrs. Longmans, publishers.

COSENTINO. Atlas of anatomy and surgical approaches in orthopaedic surgery—upper extremity.

ROBINS. Injuries and affections of the hand.

WATSON-JONES. Surgery is destined to the practice of medicine. Hunterian Oration 1959. Gift of Sir Reginald Watson-Jones.

Pathology

PEERY AND MILLER. Pathology, a dynamic introduction to medicine and surgery.

Physics

EIRICH (editor). Thermodynamics and mechanisms of polyester systems. (New York Academy of Science.) Gift of Sir Cecil Wakeley, Bt.

Physiology

BEST AND TAYLOR. Physiological basis of medical practice, 7th edition.

BROCK. Recent advances in human nutrition.

BUTLER. Pancreatic secretion after gastrectomy. Bristol M.D. thesis 1959. Typescript. Gift of the author, Mr. T. J. Butler, F.R.C.S.

CIBA. Symposium on the nature of sleep.

Plastic surgery

MCINDOE. Total reconstruction of the burned face 1958. Typescript of the late Sir Archibald McIndoe's unpublished Bradshaw lecture. Gift of Sir Cecil Wakeley, Bt.

Radiology

CRONKITE AND BOND. Radiation injury in man. 1960.

Sociology

RUBIN (editor). Culture, society, and health. (New York Academy of Science.) Gift of Sir Cecil Wakeley, Bt.

Surgery

ANANYEV (editor). New Soviet surgical apparatus and instruments and their application, translated by J. B. Elliott.

COHN. Strangulation obstruction.

FOOTE AND DINGLEY. Varicose veins, 3rd edition. Gift of Mr. R. Rowden Foot, F.R.C.S., and Mr. A. Gordon Dingley, F.R.C.S., authors.

GOLIGHER. Surgery of the anus, rectum and colon. Gift of the author, Professor J. C. Goligher, F.R.C.S.

HARDY. Surgery of the aorta.

HOWARD AND JORDAN. Surgical diseases of the pancreas.

HUGHES AND BOWERS. Traumatic lesions of the peripheral vessels.

SMITH. Progress in clinical surgery, 2nd series.

STERLING. Congenital biliary atresia. 1960.

Thyroid

PITT-RIVERS (editor). Advances in thyroid research. (Transactions of the 4th International Goitre Conference 1960.)

Urology

NEGRI. La histofisiopatologia de las vias biliares. 1941. Gift of Sir Heneage Ogilvie.

PAVEL. La vésicule biliaire et ses voies d'excrétion. 1959. Gift of the Medico-Pharmaceutical Institute, Tirgu-Mures, Rumania.

HISTORICAL COLLECTION

Historic texts

THEODORIC. Surgery (A.D. 1267), translated by E. Campbell and J. Colton. 1960. Completion: vol. 1, 1955.

AMBROISE PARÉ. Case reports and autopsy records (XVI century), translated by W. B. Hamby. 1960.

FELIX PLATTER. Beloved son Felix (Journal selections, 16th century), translated by S. Jennett.

THOMAS BARTHOLIN. On the burning of his library (1670) and On medical travel (1674) translated by C. D. O'Malley. Gift of the translator and publisher, University of Kansas.

THOMAS HOWITT, F.R.C.S. Notes of the lectures and practice of Charles Bell and Guillaume Dupuytren, 1830–38. Manuscript.

SIR FREDERICK TREVES, Bt., F.R.C.S. Reminiscences of his practice: 1, Sir John Millais; 2, Sir Henry Irving; 3, King Edward VII. Autograph manuscript. Gift of the Dowager Lady Rigby, through Sir Cecil Wakeley, Bt.

SIR GEOFFREY JEFFERSON, F.R.S., F.R.C.S. Autograph postcard from Petrograd 1916 (photocopy). Gift of Sir Harry Platt, Bt.

History of Medicine, etc.

A. V. HILL. The ethical dilemma of science and other essays.

G. LEATHERMAN. The American Dental Society of London 1908–58. Author's gift.

LLOYD AND COULTER. Medicine and the Navy, vol. 3, 1714–1815. (To be completed by a fourth volume; vols. 1–2, 1957–58, by the late John Keevil.)

Queen Square and the National Hospital. 1960.

SAUNDERS. Histories of (1) the Bristol Eye Hospital, and (2) the Royal Hospital for Sick Children, Bristol. (Pamphlets.) Gift of the Hospitals.

SEYMER. Florence Nightingale's nurses. 1960.

SOMERVILLE. The Savoy: manor, hospital, chapel.

TOVELL. References to Australia in British medical journals prior to 1880. Author's gift.

VALENTIN. Geschichte der Orthopädie. 1960.

Vienna, Accident Hospital. 25-Jahre Unfallkrankenhaus Wien, 1952. Gift of Dr. K. N. Chinoy.

Biography and Bibliography

ROBERT BOYLE: Fulton. A bibliography of the Hon. Robert Boyle, F.R.S., 2nd edition.

RICHARD CHARLES: Presentation to R. Charles, F.R.C.S.I. (with reminiscences of surgery in the first world war, etc.) Privately printed pamphlet, presented by Sir Zachary Cope.

EDWARD JENNER: Dolan. Jenner and the miracle of vaccine.

SIR GEOFFREY KEYNES: Osler Club. Tributes on his seventieth birthday, with a check list of his publications.

MARCELLO MALPIGHI: Frati. Bibliografia Malpighiana (1898), re-issued 1960.

RUDOLPH MATAS, Hon. F.R.C.S.: Cohn. Rudolph Matas, a biography. 1960.

JEAN T. NICOLESCO: Travaux scientifiques. 1959. Gift of the Medico-Pharmaceutical Institute, Tirgu-Mures, Rumania.

LOUIS PASTEUR: Nicolle. Louis Pasteur, a master of scientific enquiry.

A. I. WILLINSKY: A doctor's memoirs. Gift of Messrs. Macmillan, publishers.

HOWARD GRAY LIBRARY

CARSTEN. The ascendancy of France 1648–88. (New Cambridge Modern History, vol. 5: continuation of series.) Presented through Mr. A. Dickson Wright.

DOWN HOUSE LIBRARY

BAILEY. Handlist of the Darwin papers in the University Library, Cambridge.

ZBIOROWA. Stulecie dziela Darwin 1960. Gift of the Polish Academy of Sciences, Krakow.

DONATIONS

DURING THE LAST few weeks the following generous donations have been received:

Appeal Fund—Contributions:

£3,571 8s. 6d.	Shell Petroleum Co., Ltd. (further gift)
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£1,000	The Dulverton Trust
£350	Royal Insurance Co., Ltd. (further gift)
£105	Allied Leather Industries, Ltd.
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McIndoe Memorial Fund:

$10	Ellery C. Gay, M.D.

Voluntary subscriptions and donations by Fellows:

The following Fellows of the College have generously given a donation or undertaken to make a voluntary annual subscription under covenant:

F. Ridley, F.R.C.S. A. G. G. Robson, F.R.C.S.

PROCEEDINGS OF THE COUNCIL IN JULY

AT A MEETING of the Council on 13th July 1961, with Sir Arthur Porritt, President, in the Chair, Sir Arthur Porritt, K.C.M.G., K.C.V.O., C.B.E., F.R.C.S., was re-elected President. Professor Digby Chamberlain, F.R.C.S., and Sir Eric W. Riches, M.C., F.R.C.S., were elected Vice-Presidents.

Professor C. A. Wells, F.R.C.S., Professor R. Milnes Walker, F.R.C.S., and Professor Ian Aird, F.R.C.S., were re-admitted as members of the Council, and Mr. Norman Capener, F.R.C.S., and Mr. E. G. Muir, F.R.C.S., were admitted as members of the Council. Professor W. W. Mushin, M.R.C.S., D.A., F.F.A., was admitted as a co-opted member of the Council representing Anaesthetics.

Dr. R. A. S. Cory, O.B.E., M.R.C.S., of Nassau, Bahamas, was admitted to the Fellowship (by election).

Sir Howard Florey, P.R.S., and Professor H. Wangensteen, B.A., M.D., Ph.D., of the University of Minnesota Medical Centre, were elected Honorary Fellows of the College.

The death of Mr. R. C. Davenport, F.R.C.S. (past Member of the Court of Examiners), was recorded with deep regret.

Sir Reginald Watson-Jones, F.R.C.S., and Mr. W. A. Mill, F.R.C.S., were re-elected and Mr. A. S. Aldis, F.R.C.S., Mr. H. Daintree Johnson, F.R.C.S., and Professor H. W. Rodgers, O.B.E., F.R.C.S., were elected Members of the Court of Examiners.

The Hallett Prize was awarded to Peter Fells of the University of Cambridge and University College Hospital on the result of the last Primary Examination for the Fellowship.

Brigadier L. R. S. Macfarlane, O.B.E., M.D., was awarded the Mitchiner Medal for 1961 on the nomination of the Director General of the Army Medical Services.

It was reported that Mr. P. H. Golding-Wood, B.Sc., F.R.C.S., and Dr. Michael Honey, B.A., M.B., B.Ch., M.R.C.P., had been elected Mackenzie Mackinnon Research Fellows.

The following were appointed Sir Arthur Sims Commonwealth Travelling Professors for 1962:

(1) Professor C. H. Stuart-Harris, of the University of Sheffield, to visit Australia, New Zealand, Malaya and Singapore.

(2) Professor J. B. Kinmonth, F.R.C.S., of St. Thomas's Hospital, to visit Pakistan, India and Ceylon.

(3) Professor Walter MacKenzie, of Alberta, to visit Nigeria, the West Indies, and Britain.

Mr. F. W. Holdsworth, F.R.C.S., was appointed the Watson-Jones Lecturer for 1961.

Professors and Lecturers for the ensuing year were appointed as follows:

HUNTERIAN PROFESSORSHIPS

(*Lectures on Comparative Anatomy and other subjects, illustrated by preparations from the Hunterian Collection and other contents of the Museum, by Fellows and Members of the College.*)

G. H. MACNAB.—One lecture on a study of two hundred cases of hydrocephalus treated by insertion of the holter valve.
A. GOUREVITCH.—One lecture on the surgery of jaundice in the newborn, with special reference to congenital biliary atresia.
G. W. TAYLOR.—One lecture on arterial grafting for gangrene.
D. J. WATERSTON.—One lecture on atresia of the oesophagus.
J. H. JOHNSTON.—One lecture on vesico-ureteric reflux; its anatomical mechanism, causation, effects and treatment in the child.
N. H. ANTIA.—One lecture on reconstruction of the face in leprosy.
G. SLANEY.—One lecture on hypersensitivity granulomata and the alimentary tract.
C. Q. HENRIQUES.—One lecture on the veins of the vertebral column and their role in the spread of cancer.
A. E. FLATT.—One lecture on surgical rehabilitation of the rheumatoid hand.
G. E. HEARD.—One lecture on nerve sheath tumours and von Recklinghausen's disease of the nervous system—a clinical study.
C. SHALDON.—One lecture on dynamic aspects of portal hypertension.
D. F. N. HARRISON.—One lecture on the relationship of osteomata of the external auditory meatus to swimming.
J. B. M. ROBERTS.—One lecture on spina bifida and the urinary tract.

ARRIS AND GALE LECTURESHIPS

(*Lectures on subjects relating to Human Anatomy and Physiology.*)

A. NAYLOR. One lecture on the biophysical and biochemical basis of intervertebral disc herniation and degeneration.
D. ANNIS.—One lecture on a study of the regenerative capacity of the epithelial lining of the urinary bladder, together with evidence concerning the function of the mucosa in the reflex act of micturition.
D. B. MOFFAT.—One lecture on the embryology of the arteries of the brain.
P. G. KONSTAM.—One lecture on spinal tuberculosis in Nigeria.

BLAND-SUTTON LECTURESHIP

F. STANSFIELD.—One lecture.

ERASMUS WILSON DEMONSTRATIONSHIPS

(*Demonstrations on the Pathological contents of the Museum by the Pathology Curator or some other duly qualified person or persons.*)

L. BITENSKY.—One demonstration.
J. L. EMERY.—One demonstration.
I. FRIEDMANN.—One demonstration.
J. KOHN.—One demonstration.
L. W. PROGER.—One demonstration.
R. C. B. PUGH.—One demonstration.
A. J. M. REESE.—One demonstration.

ARNOTT DEMONSTRATIONSHIPS

(*Demonstrations on the contents of the Museum by the Conservator of the Museum or some other duly qualified person or persons.*)

G. V. R. BORN.—One demonstration.
G. W. CAUSEY.—One demonstration.
J. DOBSON.—One demonstration.
C. Q. HENRIQUES.—One demonstration.
K. B. THOMAS.—One demonstration.
D. H. TOMPSETT.—One demonstration.

Licences in Dental Surgery were granted to 40 candidates.

The following diplomas were granted, jointly with the Royal College of Physicians:

Anaesthetics (95), *Laryngology and Otology* (19), *Pathology* (6), *Psychological Medicine* (64), *Public Health* (19), *Industrial Health* (1), *Child Health* (1).

The following hospitals were recognized under paragraph 23 of the Fellowship regulations:

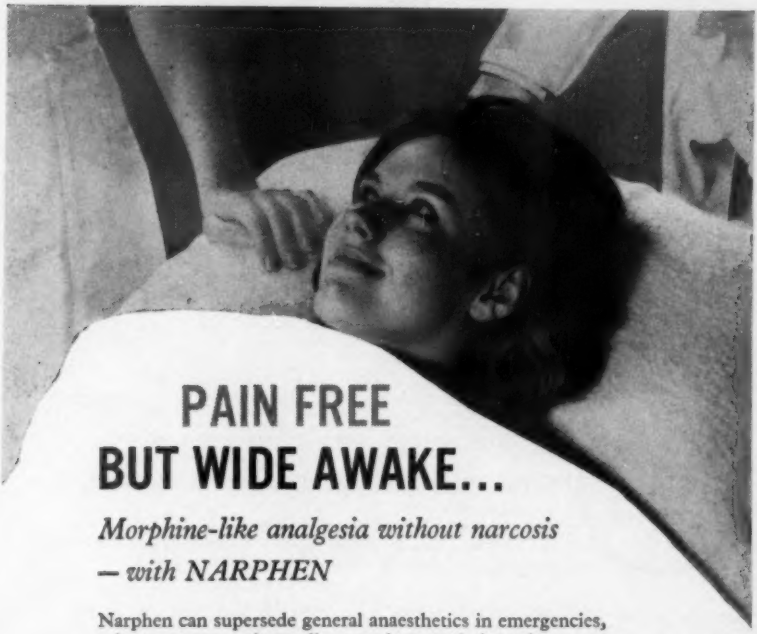
Hospitals	Posts Recognized		
	General (6 months unless otherwise stated)	Casualty (all 6 months)	Unspecified (all 6 months)
Dudley—The Guest Hospital and Eye Infirmary (additional)		S.H.O. (Cas. and Orth.)	
London—Highlands Hospital (additional)			Regr. (Orth. and Gen.)
Manchester—Crumpsall Hospital (decennial revision)	Senior Regr. (12 months) 2 Regrs. (12 months) 3 H.Os.	J.H.M.O. (Cas.)	H.O. (Orth.) S.H.O. (Urology and General) S.H.O. (Neuro-surgery) Regr. (Neuro-surgery) (or 2nd S.H.O.)
Manchester—Northern Hospital (additional)	S.H.O. H.O. (Pre-registration)		
Mitcham—Wilson Hospital		Cas. Offr.	
Pinderfields—General Hospital (additional)	Senior H.S. 2nd H.S.		
Neath—General Hospital (additional)		S.H.O. (Traumatic and Cas.)	
Swansea—General Hospital (additional)		J.H.M.O.	

DIARY FOR AUGUST

Tues.	1		Museum and Library closed this month.
Thurs.	3	2.00	Ordinary Council.
Sat.	5		College closed.
Mon.	7		College closed.

DIARY FOR SEPTEMBER

Wed.	6		College closed. Staff outing.
Wed.	13		Primary F.D.S., Second L.D.S., Pre-Medical and D.C.H. Examinations begin.
Mon.	18		First Membership Examination begins.
Tues.	19		Primary F.F.A. Examination begins.
Wed.	20		D. Orth. Examination begins.
Fri.	22	5.00	Board of Faculty of Dental Surgery.
Tues.	26		Final Membership Examination begins.



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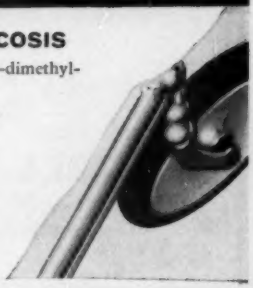

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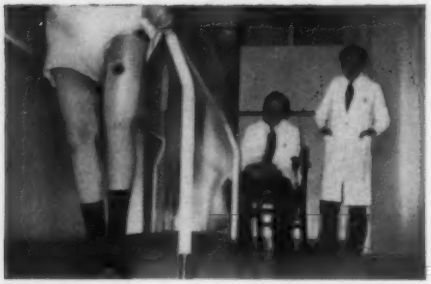

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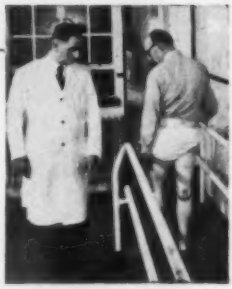

Fitting

Hanger take an enormous amount of care in the fitting of legs; preliminary fittings are made before the leg is completed so that it can be finished to the amputee's individual anatomical requirements. The illustration (right) shows an amputee walking on a newly fitted leg with the help of an experienced limb-fitter in one of our 28 branches in the United Kingdom.

Rehabilitation

After fitting, amputees enjoy all the rehabilitation and servicing facilities offered by the Hanger organisation — the largest of its kind in Britain. The ex-naval petty officer shown in the illustration is fitted with two below-knee amputation Hanger legs and leads an absolutely normal life.

(left) A light metal leg for below-knee amputation. Hanger make a full range of artificial legs both for amputations and congenital deformities, and can also supply legs for growing children. Please write for further details.

J. E. HANGER & CO. LTD Queen Mary's Hospital, Roehampton, London S.W.15

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FIGHT CANCER

WITH A WILL!

When you are consulted about bequests for Medical work which deserves support, please remember the part played by the Imperial Cancer Research Fund. It receives no official grant and is entirely supported by voluntary donation and bequest. Pioneer in the field of cancer research, this organisation was founded by the Royal Colleges of Physicians and Surgeons to undertake this vital work in its own laboratories. Please help us when you can!

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Write for further information to A. Dickson-Wright Esq., M.S., F.R.C.S.
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